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Symposium On The Circulation

William Harvey and His Times

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Introduction

This issue of the Manitoba Medical Review signalizes the tercentenary of the passing of William Harvey; for according to Aubrey, "On the third day of June, 1657, about ten in the morning, Harvey, then in his eightieth year, on attempting to speak found that he had lost the power of utterance . . . by and by in the evening of the day in which he was smitten he died, the palsy giving him an easy passport."

Harvey is often called "the patron saint of Physiology;" in fact he was Lumleian Lecturer on Anatomy and Surgery to the College of Physicians of London. Moreover, his great work he entitled Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus—not a physiological treatise, but an anatomical treatise; and the viewpoint of modern Anatomy could scarcely find better expression than in the title chosen by Harvey. His own feeling on this point is made clear by his designation of himself on the title-page as "Physician to the King, and Professor of Anatomy to the College of Physicians of London."

But Harvey has another claim to recognition as an anatomist; had he never demonstrated that the blood circulates he would still enjoy an honoured place in anatomical history. His great fame in connection with the circulation has caused his merits as an embryologist to be commonly overlooked; I imagine that many doctors would be puzzled if asked, For what is Harvey famous, apart from the circulation?

Harvey is commonly referred to as the discoverer of the circulation of the blood. It seems to me, however, that scientific knowledge is not discovered—it is made, in the sense that music is composed and literature is written. This implies that scientific research is a creative art, and that Harvey belongs in the goodly company of his contemporaries, Shakespeare, Rubens and Monteverde; like them, he is a landmark in the history of his art.

His Life and Times

Though a quiet little man (albeit somewhat irascible), Harvey like all artists expressed something of the times in which he lived, and stirring times they were.

Born in Folkestone, Kent on April 1, 1578, Harvey grew to manhood "in good Queen Bess's glorious days." At the time of his birth, Shakespeare and Galileo were boys of fourteen, and Sir Francis Drake was making the first voyage round the world by an Englishman. The years up to Harvey's entering Cambridge saw the founding of the British Empire; for in 1583 Sir Humphrey Gilbert formally laid claim to Newfoundland, which thus attained her proud status as "Britain's oldest colony;" and two years later Sir Walter Raleigh founded his first colony in Virginia. In 1587 Mary, Queen of Scots, laid her unhappy head upon the Elizabethan block; and the very next year saw the débacle of the Invincible Armada.

In 1593 Harvey entered Gonville and Caius College, Cambridge. John Caius (1510-1573) had studied Anatomy under the great Vesalius in Padua, and had introduced into England Vesalius' revolutionary teaching methods. It was while Harvey was a medical student at Cambridge, at the end of the sixteenth century, that the Medical Faculty of the University of Mexico was established.

After graduating in Arts at Cambridge in 1597, Harvey, following the example of Caius, proceeded to Padua to study Medicine, remaining until 1602. As we shall see, the importance of the influences bearing upon Harvey in Padua can hardly be exaggerated. Two sharply contrasting events that occurred in the year 1600 illustrate the change that was taking place. In Rome Giordano Bruno for his scientific and theological indiscretions was "punished with all possible clemency, and without shedding of blood," according to the official formula—i.e., by being burned at the stake. In England Dr. William Gilbert of Colchester, physician to Queen Elizabeth, published his De Magnete, etc., the first major original contribution to Science published in that country, and one of the foundations of our knowledge of electricity. In the same year the East India Company was formed; Britain was expanding in another direction.

Upon graduating in Medicine in 1602, Harvey returned to England; until about 1630 he practised in London (which suffered several outbreaks of the plague) and pursued his scientific investigations. In 1603 Queen Elizabeth died, and was succeeded by the son of Mary, Queen of Scots, James VI of Scotland and I of England. The next year Harvey married the daughter of a court physician; his wife predeceased him, and they had no children. In 1605 Bacon's Advancement of

Learning was published; in 1606 Rembrandt was born; and in 1607 Harvey was elected a Fellow of the College of Physicians. In 1608 Milton was born, and Samuel de Champlain founded Quebec city; the next year he discovered Lake Champlain —the year in which Galileo invented the telescope, and Harvey became physician to St. Bartholomew's Hospital. In 1610 Henry Hudson perished in the waters of the great bay that bears his name; 1611 is memorable for the appearance of the Authorized (King James) Version of the Bible; and in 1614 John Napier of Merchiston (near Edinburgh) gave to the scientific world the gift of logarithms.

In 1615 Harvey was appointed, as already mentioned, Lumleian Lecturer on Anatomy and Surgery to the College of Physicians; the next year, in his first course of lectures (delivered in Latin, of course), he expounded his views on the circulation of the blood; this was the year of Shakespeare's death. In 1618 Harvey was appointed physician to James I. Another famous patient was the Lord Chancellor, Francis Bacon (Lord Verulam), whose Novum Organum appeared in 1620, the year of the Pilgrim Fathers. Harvey's dry comment on Bacon is justly famous: He writes philosophy like a Lord Chancellor. In 1621 Burton's Anatomy of Melancholy appeared; in 1625 Sydenham was born. Upon the death of James I in 1625 Harvey became physician to his successor, Charles I. Incredible though it seem, Harvey was actually suspected of responsibility for James's death; fortunately he was completely exonerated.

In 1628 Harvey's De Motu Cordis was published, when he was fifty, and some twelve years after his first lecture upon it. During the 1630's, consequent upon his popularity with the monarch and the court, Harvey did considerable travelling. In 1634 came the affair of the witches (see below): and in the following year he made his "Anatomical Examination of the Body of Thomas Parr, Who Died at the Age of One Hundred and Fifty-two Years." In 1636 Harvard College was founded; in the next year there appeared Descartes' "Discours de la Méthode." In 1639 the first hospital in Canada was founded, the Hôtel Dieu du Précieux Sang in Quebec; and 1641 saw the beginning of the city of Montreal.

In 1642 the Civil War broke out in England, and Harvey's London quarters were looted, much valuable scientific material being destroyed; in the same year he was with the King at the battle of Edgehill. 1642 is also notable for the discovery of New Zealand and Tasmania by Tasman; the death of Galileo; the birth of Newton; and the publication of Sir Thomas Browne's Religio Medici, affectionately called by Sir William Osler: Comes viae vitaeque (my companion on the road of life). In 1643 the barometer was invented by Torricelli, a pupil of Galileo; and Harvey retired from Barts. In 1645 he was made Warden of Merton College, Oxford; but the very next year he had to flee from Oxford with the King. Suffering from gout, Harvey retired from medical life in 1648, at the age of seventy; thereafter he lived quietly with one or other of his surviving brothers in or near London. In 1649 Charles I was beheaded, and Oliver Cromwell became Protector.

In 1650 Harvey was visited by his friend Sir George Ent, who fortunately succeeded in persuading him to consent to the publication of his work on Embryology, which appeared in the following year under the title: Exercitationes Anatomicae de Generatione Animalium (Anatomical Discourses on the Reproduction of Animals). In 1654 Harvey was chosen President of the College of Physicians, but he declined office because of age and infirmity. In 1656 he resigned from his anatomical lectureship, which he had held for forty years. Then, we are informed, "Of William Harvey, the most fortunate anatomist, the blood ceased to move on the third day of the Ides of June, in the year 1657, the continuous movement of which in all men, moreover, he had most truly asserted."

One mystery concerning Harvey remains unsolved to this day, so far as I know. With all his scientific and medical eminence, and considering his position at court and royal favour, why was no knighthood or other title bestowed upon him? That some doctors were so honoured in those days is attested by the knighthood conferred by Charles II upon his physician Charles Scarborough, mentioned in Harvey's will as "my loving friend." Did Harvey decline?

Paduan Influences

In the history of our civilization Science has arisen twice: in ancient times, and again in modern times. Ancient Science came to an end with the death of Galen in 200 A.D.; its most influential figures were Hippocrates, Aristotle and Galen. Modern Science dates from the Renascence. What was born again at that wonderful time? Fundamentally, the ancient Greek spirit of free critical inquiry. This revival was centred in northern Italy, particularly in Padua. So far as Science is concerned, the important date is 1543, which saw the publication of two great books, one on Astronomy, the other on Anatomy — one on the Macrocosm, the other on the Microcosm. The De Revolutionibus Orbium Coelestium (On the Revolutions of the Heavenly Bodies) by the Polish doctor Copernicus undermined faith in the writings of the ancients as infallible sources of scientific knowledge. The De Humani Corporis Fabrica (On the Structure of the Human Body) by the Paduan anatomist Vesalius blazed in more positive fashion the modern trail of scientific knowledge, the toilsome trail of observation. The immediate successors of Vesalius in the chair of Anatomy at Padua were Columbus, Fallopius and Fabricius, the latter the teacher of Harvey.

"The celebrated Hieronymus Fabricius ab Aquapendente, a most skilful anatomist and venerable old man," as Harvey calls him, was born about 1533, succeeded Fallopius in 1565, retired in 1613, and died in 1619; he taught Anatomy for over half a century. His patients included Galileo, of whom more presently. Fabricius published a number of anatomical treatises, three of which interest us particularly because of their influence on Harvey. One deals with the cardiovascular system, the other two with Embryology; Harvey followed his master and excelled him in both fields. Happily, these publications are now available in English: De Venarum Ostiolis (On the Valves of Veins, 1603) translated (1933) by K. J. Franklin of Oxford (now of London); and De Formato Foetu (On the Formed Foetus, 1604) and De Formatione Ovi et Pulli (On the Formation of the Egg and of the Hen, 1621), both translated (1942) by H. B. Adelmann of Cornell. From these we can glean a fair idea of Harvey's indebtedness to Fabricius. In addition to cardiovascular and embryological problems, Harvey also acquired from Fabricius an appreciation of the importance of Comparative Anatomy in connection therewith.

For historical and political reasons that need not be discussed here, the fires of the Renascence burned brightly in Padua: critical inquiry, first-hand observations and original ideas were less unwelcome there than elsewhere at that time. But it should not be forgotten that opposition drove her two brightest scientific intellects to shake the dust of Padua from off their feet, Vesalius in 1544, Galileo in 1610. Our story is laid in the transition period between mediaeval and modern times.

During Harvey's stay in Padua its greatest intellect and most potent scientific influence was Galileo. Born in 1564, the year in which Vesalius and Michaelangelo died and Shakespeare was born, Galileo was professor of Mathematics, first at Pisa, then at Padua from 1592 to 1610. His unhappy encounters with the Inquisition (after his departure from Padua) need not concern us here; suffice it to say that, like Bruno, Galileo had a most unfortunate propensity toward indiscretion; but though treated severely he was spared such cruelty as Bruno suffered. When he was old and blind he enjoyed a visit from Milton; he died in 1642.

The massive contributions of Galileo cannot even be summarized here; a few general statements must suffice. Men have experimented throughout the ages, and a few performers of experiments, like Archimedes, occurred in the ancient world. But the first systematic experimenter — i.e., the first to execute series of experiments designed to solve specific problems—was Galen, who was thus not only a physician, but an experimental biologist. Galileo was the first systematic experimenter in any science since Galen. But he introduced something new into Science, quantitative experimentation, together with a fruitful alternation of inductive and deductive reasoning. This method was perfected by Sir Isaac Newton, who was born in the year of Galileo's death; by it all scientific advances were made until the twentieth century, with its Uncertainty, its Probability, and its Relativity.

The first to apply Galileo's quantitative experimental methods to living things was a Paduan colleague, Sanctorius (1531-1636), who was professor of Medicine, and studied metabolism, fever, and other matters. But by far the most important of Galileo's immediate biological followers was Harvey. It was largely by Galileo's methods that he demonstrated that the blood circulates. This at once raises the question, What was the relationship between Galileo and Harvey in Padua? In this connection I am going to offer two quotations. The first was written by Sir George Newman.

"For eighteen years the influence of Galileo permeated the first medical school of the age and left its ineffaceable mark upon the growing science of all living things. Harvey himself must often have listened to the Professor of Mathematics, for the aula magna and the anatomical theatre adjoined each other, and Galileo's popular lectures were the talk of the University." The second quotation was written by myself some years ago.

"Though Harvey refers to Fabricius, he makes no mention of Galileo. This suggests that Harvey himself did not recognize Galileo's influence over him; that influence may have been exerted not personally (as was Fabricius'), but indirectly. So powerful an intellect as Galileo's must have influenced all Padua (except, apparently, Fabricius!); hence Harvey may have thought of it (if he thought of it at all) as simply a Paduan influence, overlooking its personal origin."

It is interesting that Galileo's methods were speedily transferred to England, where they were applied first to Biology by Harvey and later to physics by Newton.

The Circulation

Accounts of Harvey's specific observations, experiments and conclusions are so readily available in numerous publications (a few of which are listed below) that only some general remarks are offered here.

Harvey's precursors in this field are discussed specially by Flourens and by Bayon. Suffice it to say that whilst the passage of blood through the lungs may have been divined by Servetus and demonstrated by Columbus, Harvey does seem to have been the first to infer that the same blood that passes through the lungs also circulates throughout the rest of the body, and he was certainly the first to demonstrate experimentally the truth of this idea. He also elucidated a large number of points in the Dynamic Anatomy of the cardiovascular system, such as the significance of the valves, details of the heart's action, the relationship of cardiac systole to the movement of the blood and to the pulse wave, and the passage of all the blood through the lungs.

He did all this by a combination of the comparative and experimental methods of Fabricius and the quantitative and logical methods of Galileo. Daremberg (quoted by Wyatt) remarked: "Harvey considers a long time and he finishes by seeing; he makes few experiments, but they are decisive; he uses arguments, but they are conclusive." After all, knowledge is made not by methods (as Bacon would have it), but by men and women. Harvey had the creative artistic gift that transcends method and leads its possessor to his goal. Nevertheless, the goal is attained by the artistic use of methods, and experience of methods favours, in some people, the development of innate talents.

The reception accorded to Harvey's work, as to that of Lister, was mixed. Aubrey wrote: "I have heard him say that after his booke of the Circulation of the Blood came out he fell mightily in his practice, and 'twas believed by the vulgar that he was crack-brained, and all the physitians were against him." His attitude toward his professional opponents resembled that of Lister; and both had the satisfaction of seeing their work widely accepted during their lifetime. Notwithstanding this, it was many a long year before the doctrine of the circulation exerted its full effect upon the practice of Medicine.

In Harvey's concept of the circulation there was one anatomical missing link. He postulated a series of minute "porosities" connecting arteries and veins; failing to use even such microscopes as were then available, he could not demonstrate these communications. It is an interesting fact that the man who first demonstrated them in the lungs, Marcello Malpighi (1628-1694), professor of Medicine at Bologna, was born in the very year of the publication of Harvey's treatise, 1628; he described the capillaries in his De Pulmonibus, 1661.

A post-script to Harvey's work on the circulation may serve as a a corrective to excessive hero-worship. In 1622 Aselli described the lacteals, and in 1651 Pecquet described the receptaculum chyli and the thoracic duct, and the relationship thereto of the lacteals; the ordinary lymphatic vessels were discovered by Joyliffe in 1652 and by Rudbeck in 1653. Yet in 1652 Harvey wrote a letter stating that he had seen the lacteals before Aselli, but denying the significance attached to them by Pecquet. Thus the "discoverer of the circulation" failed to recognize the discovery of the lymphatic system when it was drawn to his attention!

Embryology

Harvey was the most distinguished embryologist since Aristotle, the only outstanding figures between them being Coiter and Fabricius. But the embryological techniques of Fabricius and Harvey were hardly much advanced over those of Aristotle, their views were based on his, and it was much more difficult than in the case of the circulation to adduce new observations to serve as a basis for advanced ideas.

It is interesting to survey what Harvey did accomplish under these circumstances; since this is much less widely known than his work on the circulation, I shall refer to it under the following headings: development of the chick, mammalian embryology, and general embryology.

Concerning the development of the chick, and referring to Harvey's Oxford period, Aubrey recollects: "I remember that he came often to Trin. Coll. to one George Bathurst, B.D. who kept a hen in his chamber to hatch egges, which they did dayly open to discerne the progress and way of generation." All honour to George Bathurst, B.D. It may be said that, using merely a hand lens, Harvey extended our knowledge of the development of the chick almost as far as possible under such technical limitation. Naturally he was greatly interested in the development of the heart and vessels, so readily observed in the chick.

Harvey's opportunities for investigating mammalian embryology were unique, as he himself relates.

"It was customary with his Serene Majesty, King Charles . . . to take the diversion of hunting almost every week . . . and no prince in the world had greater herds of deer . . . This gave me an opportunity of dissecting numbers of these animals almost every day, during the whole of the season . . . because the great prince, whose physician I was, besides taking much pleasure in such inquiries and not disdaining to bear witness to my discoveries, was pleased in his kindness and munificence to order me an abundant supply of these animals, and repeated opportunities of examining their bodies."

As we might expect, macroscopic observations upon mammalian embryos and their membranes were much more difficult than those upon hen's eggs. Although this section of his work abounds with specific observations, these referred less to organogenesis than to problems of general embryology.

These problems Harvey acquired largely from Fabricius, and indirectly from Aristotle. But, lacking microscopic technique, Harvey remained unaware of ovum, spermatozoon, fertilization, and much more that is common knowledge to us; thus he was free to indulge in mediaeval speculations, couched in Aristotelian terms and concepts, and unchecked by relevant observations.

The frontispiece of his book shows Zeus releasing living things from an egg, on which is written: Ex ovo omnia. Nothing is easier, or more fallacious, than to imagine that this meant the same to Harvey that it does to us—he had no notion of the ovum as we know it, especially the mammalian ovum. In certain instances, such as parasites, he naturally found the idea of spontaneous generation hard to give up.

Harvey supported the doctrine of epigenesis, the gradual development of anatomical structures out of unformed material. Though nearer our modern viewpoint than the rival doctrine of preformation, Harvey's "epigenesis" corresponded more to Aristotle's idea of the developmental process than to ours.

We may sum up the situation by stating that the De Generatione was the finest work on Embryology that had appeared thus far.

Parr and Parrot

This heading refers to two necropsies of which we have Harvey's own reports; a third is mentioned in the next section. As already noted, in 1635 Harvey performed a necropsy upon the body of "Thomas Parr, a poor countryman . . . (who) died on the 14th of November, in the year of grace 1635, after having lived one hundred and fifty-two years and nine months, and survived nine princes." The necropsy was performed "by command of his Majesty, several of whose principal physicians were present." The report, however, was not published until twelve years after Harvey's death. It reveals him as a matter-of-fact physician. No cause of death being obvious, Harvey expresses some opinions about this.

The other necropsy was on Mrs. Harvey's parrot, of which (it is easy to read between the lines) he was as fond as she was. After describing its attractive and endearing ways, Harvey continues:

"I always looked upon the creature as a male on account of its skill in talking and singing . . . until . . . the parrot, which had lived for so many years in health, fell sick, and by and by being seized with repeated attacks of convulsions, died, to our great sorrow, in its mistress's lap, where it had so often loved to lie. On making a post-mortem examination to discover the cause of death I found an almost complete egg in its oviduct, but it was addled."

The Witches

Belief in witches was commonplace in the age in which Harvey lived. Even Sir Thomas Browne gave evidence that helped to convict two such wretches in 1664; and the affair of the witches of Salem occurred in 1692, long after Harvey's death.

In 1634 a boy who had played truant from school told a tale about witches and witchcraft that caused seven Lancashire women to be thrown into jail, where three of them died. The survivors were brought to London, where the King ordered them to be examined for stigmata (supernumerary nipples and the like) by seven surgeons and ten midwives. Fortunately for the women, the King also ordered that the surgeons and midwives receive their instructions from Harvey. Though we know no details, we do know that the women were exonerated; considering the current feeling, doubtless they had to thank Harvey for that.

This is rendered the more likely by a story that has come down to us of Harvey's dealings with another witch and her "familiar," a semi-domesticated toad. Having heard of this old woman's reputation, Harvey visited her to ascertain the facts. He must have found the toad particularly interesting; for he got rid of the woman on some pretext, and proceeded to perform a necropsy on the animal! He concluded that this toad "no ways differed from other toades." He must have considered this significant evidence; for a contemporary justice of the peace relates that when he asked Harvey whether there was any such thing as witchcraft, "Hee told mee, he believed there was not."

Conclusion

To appreciate Harvey and his work, we must not imagine that the scientific Renascence was complete in his day, or that he was a modern scientist and physician. He was a transitional figure, living in a transitional age, and contributing mightily to the transition. The following quotation from Garrison illustrates the viewpoint of the present essay.

"Dr. John J. Abel is quite right when he says that 'there should be in research work a cultural character, an artistic quality, elements that give to painting, music and poetry their high place in the life of man.' The real student of medical history will see his Hippocrates as he does his Homer, his Harvey as he does his Shakespeare, his Sydenham as he does his Milton."

Finally, as we take affectionate leave of our noble little man, let us have in mind the concluding words of the dedication of his great work.

"I avow myself the partisan of truth alone; and I can indeed say that I have used all my endeavours, bestowed all my pains on an attempt to produce something that should be agreeable to the good, profitable to the learned, and useful to letters.

Farewell, most worthy Doctors,
And think kindly of your Anatomist,
William Harvey."

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The Pulmonary Circulation

Its Role in Cardiorespiratory Disease

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Introduction

The pulmonary circulation, in the anatomical sense of the term, comprises the route taken by the blood from the right ventricle, via the pulmonary arteries, capillaries and veins, through the lungs to the left atrium. The bronchial circulation, however, deriving from branches of the aorta, belongs to the systemic circulation. In view of the close anatomical connections between pulmonary and bronchial circulations in the normal and the diseased lung it is felt that it is desirable to include the bronchial circulation in the present discussion. Furthermore, although not infrequent observers have called attention to changes in the bronchial circulation in diseases affecting the lungs and the pulmonary blood flow, it seems that too little attention may have been paid to the fact that the lungs have a double blood supply.

At this time, when tribute is being paid to the contributions of William Harvey and to his influence on the study of the circulation in health and disease, it is felt that an historical approach to the problem is the most appropriate. We have therefore attempted to indicate the trends in the development of our present knowledge of the subject and to give some indication of the practical applications of physiological theories and experimental methods in the clinical study of cardiorespiratory disease.

Early History

The first account of the pulmonary circulation appears to be that of Ibn an-Nafis who lived in Cairo about 1210-1288. He clearly described the right and left hearts and appreciated that the venous blood passed from the right ventricle, via the pulmonary artery, to the lungs "in order to expand its volume and to be mixed with air so that its fumes and part may be clarified." The blood then returned via the pulmonary vein to the left side of the heart.

Thereafter these writings were lost sight of and it was not until 1553 that Michael Servetus published an account of the pulmonary circulation explaining how the blood passed through the lungs from the right heart to the left. It appears that the description of Servetus was made independently of any knowledge of Ibn an-Nafis, and it is interesting to note that it appeared only as an argument by analogy in the course of a lengthy theological treatise.

In the years that followed a gradual growth of knowledge of the heart and circulation developed. Colombo in 1559 included a description of the pulmonary circulation in his work, but considered that the liver was the centre of the circulatory system. Cesalpino in 1593 published an account of the circulation which preceded that of Harvey by 57 years. While this description was in all essentials similar to that of Harvey, it was buried between several other publications on diverse topics and did not attract the attention it deserved.

On his appointment as Lumleian Lecturer by the College of Physicians of London, Harvey delivered his first lectures in April of 1616. In his manuscript notes it is evident that he had a clear idea of the circulation of the blood at that time. He writes "it is plain from the structure of the heart that the blood is passed continuously through the lungs to the aorta as by the two clacks of a water bellows . . . it follows that the movement of the blood is constantly in a circle."

In 1628 the appearance of "DeMotu Cordis" finally crystallised the concept of the circulation, including the circulation of blood through the lungs.

With the discovery of the capillaries by Malpighi in 1660, the anatomical continuity of the circulatory system, postulated by Harvey on the basis of his observations, was completed.

It is perhaps not so widely known that in the same year Malpighi described the vesicular structure of the lung. Before Malpighi, following Vesalius in 1543, it had been considered that the blood and air mingled in the lungs for the purposes of aeration and purification of the blood. Malpighi proved, by the injection of mercury, that the blood and air did not intermingle and described the lobular structure of the lung as consisting of vesicular spaces with walls of delicate membranes which communicated with each other and with the bronchi.

Following Harvey, an increasing knowledge of the circulation developed and, even in the earliest times, the trends that were to be maintained almost to the present day became apparent in historical reviews of the literature of the period. The systemic circulation was susceptible to direct clinical observation and, by the commencement of the 19th century, clinical cardiology was beginning to assume something of a modern appearance. In addition, the invention of the stethoscope by Laennec in 1816 led to further advances in the study of both cardiac and respiratory disease. The pulmonary circulation, however, could not be studied directly and apparently received only passing mention in the literature of the time. As far as can be determined no attempts were made to ascribe any diseased process to primary changes in the pulmonary circulation.

While knowledge of the systemic and, to a lesser extent, the pulmonary circulation had been developing, and bronchial circulation was described as a separate circulation to the lungs. According to Cockett and Vass (1950), Marchetti first described the bronchial arteries in 1654. W. S. Miller (1937) agrees but points out that Friedrich

Ruysch, in 1732, claimed to be the first to have observed these vessels. In this early period, workers in this field considered that the bronchial arteries anastomosed directly with the pulmonary arteries. This view was not challenged until Guilliot in 1835 and 1845 claimed, on the basis of injection studies, that no such connections existed.

Nineteenth Century

Although the basic anatomical facts concerning the pulmonary and the bronchial vessels were established by the beginning of the nineteenth century, there was little or no detailed knowledge of their finer ramifications. Anatomically, attention became focused on the study of the minute anatomy of the lungs with special reference to the relationships and structure of the terminal respiratory portions. As far as can be determined, no work devoted entirely to a detailed study of the bronchial tree and its relation to the vessels in the lungs appeared in English until Ewart (1888) published his treatise. He demonstrated the segmental arrangement of the bronchi and showed how the pulmonary arteries followed the bronchi to the peripheral parts of the lobules.

At about this time W. S. Miller commenced his extensive studies of the minute anatomy of the human lungs. His many contributions are summarised in his excellent monograph (Miller, 1937) and provide the basis for the modern nomenclature of the distal respiratory units. Miller extended the work of Ewart and confirmed the intimate relationship of the terminal pulmonary arteries and arterioles to the terminal bronchi, respiratory bronchi and alveolar ducts. Miller gave a detailed account of the bronchial circulation and considered it responsible for the supply of the bronchial walls and also of the vasa vasorum of the pulmonary arteries down to the level of the respiratory bronchi. At this level, the bronchial arteries terminated by dividing into a capillary leash which quickly merged with the capillary network of pulmonary arterial derivation. Miller denied the existence of anastomoses between the pulmonary and bronchial arterial systems, except at capillary level.

Physiological advances in the study of respiration and the pulmonary circulation in the 19th century were, perhaps, more striking than those in the anatomical field. It must be remembered that the true significance of the respiration was scarcely known and it was only in 1777 that Lavoisier, following on his basic chemical discovery of the nature of combustion, showed that, in respiration, oxygen was removed from the inspired air and that carbon dioxide was added. Even so, it was not till 1845 that Mayer put forward the theory that oxidation was the ultimate source of all energy involved in animal movements. From about this time knowledge advanced fairly rapidly.

With the invention of the kymograph by Ludwig in 1847 and of the spirometer by Hutchinson in 1849 the foundations of spirometry were laid and functional study of respiratory physiology and pathology commenced. However, direct study of the pulmonary circulation in man was still impossible and from this point until fairly recent times our knowledge of pulmonary haemodynamics has been derived largely from animal experiments.

Two early experiments exemplify the type of approach which the circumstances dictated. According to Gairdner (1851), Poiseuville, while investigating circulatory dynamics, studied the effect of distension of the lung on the rate of pulmonary blood flow. He employed an isolated lung and perfused fluid through it using a constant pressure. He found that the rate of flow was lowest when the lung was collapsed and increased when the lung was normally expanded. When overdistension was produced the rate of flow was diminished. Since then this view has been generally accepted and numerous workers have repeated Poiseville's work using either isolated lungs or more complicated perfusion methods with the chest open or closed. The second type of experiment was the first recording of the pressure in the pulmonary artery by Beutner in 1852. According to Tigerstedt (1923) this work was performed under the guidance and stimulation of Carl Ludwig and, characteristically, published under the name of his pupil.

Since these original experiments, a large volume of work developed on the dynamics and relationships of the pulmonary and bronchial circulations. The techniques became more complicated and the methods of recording more refined, but in spite of the fact that the work was necessarily confined largely to animals, much important knowledge accumulated.

In the fields of pathology and medicine it appears that advances were less striking. From the pathological point of view, work related to the pulmonary circulation developed along two main lines. In the first instance, under the influence of Virchow, detailed studies were made of the pathological changes in the lungs in cases of obvious circulatory disturbance. The phenomena of acute and chronic congestion of the lungs in cases of cardiac failure soon became familiar. At the same time the process and nature of pulmonary infarction, thrombosis and embolism were also extensively studied. In this connection it is of interest to note that Virchow (1847 and 1851), in demonstrating that pulmonary infarction resulted from pulmonary arterial obstruction and not from rupture of the artery, pointed out that, in experimental animals, large branches of the pulmonary artery could be blocked without producing infarction. He explained this on the basis of anastomoses between the pulmonary and bronchial systems which provided enough blood to keep the part alive. The interest in this observation lies in the fact that it

is one of the few instances in the literature, until recent times, where the bronchial circulation has been considered to play a significant functional role in a pathological process.

In other respects the pulmonary circulation received relatively scant pathological attention. Developments in the knowledge of pathological processes in the lungs occurred, but, in general, the pulmonary vessels were mentioned only incidentally in the course of description of primary lung pathology. In fact it is only in relation to the topic of pulmonary emphysema that the effects of pulmonary disease on the blood flow through the lungs received significant mention.

When Laennec (1819) originally described the gross anatomical and clinical features of emphysema he stated that the resistance afforded by the distended lungs caused cardiac embarrassment, leading to either dilatation or hypertrophy of the heart, but he did not specifically state that disproportionate right ventricular hypertrophy was the result and that the cause of this was the widespread destruction of capillary and vascular bed in the course of the emphysematous destruction of lung tissue. It became established that, when sufficient destruction of vessels had occurred, right ventricular hypertrophy resulted. It is fair to say that that is the present day view.

The study of emphysema provides the first example of a primary pathological process in the lungs upsetting the dynamics of pulmonary blood flow to such an extent that right ventricular hypertrophy and eventual cardiac failure could result.

In the meantime systemic arteriosclerosis was being actively studied and pulmonary arteriosclerosis had been recognized by Brisson in 1803. However, sclerosis of the pulmonary arteries was considered to be rare and was most frequently found in association with either mitral heart disease or pulmonary emphysema. It became accepted that pulmonary arteriosclerosis was a secondary phenomenon and, by implication, that raised pulmonary artery pressure was a significant causative factor. Cases of "primary" pulmonary arteriosclerosis were first described in the German literature in 1891 and 1892 by Romberg and Aust respectively. But, in general, pulmonary arterial disease was considered to occur most commonly as a secondary phenomenon in the course of cardiac or pulmonary disease.

In summary, the 19th century saw the development of a large body of pathological knowledge which touched on the pulmonary and bronchial circulations but indicated that the lung vessels themselves were seldom the seat of primary pathological changes. It was recognized that cardiac failure influenced the pulmonary vessels through the mechanisms of acute oedema of the lungs, chronic passive congestion and pulmonary thrombo-embolism and that pulmonary disease could influence the heart and cause right ventricular hypertrophy with ultimate failure.

It should be noted, however, that up to this time the concept of "pulmonary heart disease" was limited fairly sharply to the cardiac changes secondary to pulmonary emphysema, in short, "emphysema heart." Standard works on internal medicine and pathology published at the end of the 19th century, apart from "emphysema heart," make little or no mention of pulmonary heart disease or of diseases of the pulmonary vessels.

Twentieth Century

Early Work

The earlier decades of the present century saw little advance in our knowledge of the anatomy of the pulmonary circulation. On the one hand, W. S. Miller continued his studies and there was available a precise body of knowledge concerning the finer ramificatcn of the pulmonary vessels in relation to the bronchial and respiratory portions of the lungs. But there was little practical use to which this type of information could be put. However, the development of bronchoscopy, largely under the influence of Jackson, led to the adoption of a precise nomenclature of the bronchial segments which consolidated the work of Ewart in the previous century. Somewhat later, mainly in the late thirties, the growth of thoracic surgery demanded a detailed knowledge of the segmental anatomy of the lungs and this led to detailed publications such as that of Boyden (1943) in which the relations of bronchi and blood vessels were clearly defined.

Pathologically, especially in the earlier decades of the twentieth century, the main advances came as a logical sequence to the development of histopathology and bacteriology in the nineteenth century and were more concerned with the nature and pathogenesis of diseases of the lungs, heart and vessels than with the functional effects which these diseases produced on the pulmonary circulation. Primary pathology in the pulmonary vessels appeared to be uncommon and few direct studies of these vessels were made. In the English language the most comprehensive is that of Brenner (1935) who reviewed the subject and added the details of his own carefully studied material. He pointed out the rarity of primary pulmonary arteriosclerosis. Otherwise, he was impressed by the frequency with which sclerotic changes were found in the pulmonary vessels, but considered that these changes, unlike similar lesions in the systemic circulation, were attended by remarkably little functional upset. Since then more direct methods of investigating the pulmonary circulation have suggested an increasing functional significance for the sclerotic changes described so admirably by Brenner. Meanwhile, as Curator of the Medical Museum at McGill University, Maude Abbott was amassing the material which, in her hands, established her as the leading authority on congenital diseases of the heart. Her contributions appeared to culminate in the publication of her classic atlas (Abbott 1936). However, the development of

cardiac surgery in the post-war years lent new significance to the work of Maude Abbott, and there can be little doubt her descriptions of anatomical anomalies and resultant functional derangements provided basic information for the pioneer work on the surgery of congenital cardiac disorders. The bearing of this work on the pulmonary circulation is obvious in conditions such as pulmonary stenosis, patent ductus arteriosus and ventricular septal defects where both the total blood flow and the pressure relationships in the pulmonary circulation are altered. However, it must be remembered that changes in the haemodynamics of the pulmonary circulation can produce secondary changes in the lung vessels themselves which may influence the results of surgical treatment. One concrete example of such changes is the enlargement of the bronchial vessels in congenital pulmonary stenosis as first reported by Christellev (1917).

Later, with the introduction of plastic injection masses, several groups of workers have studied the changes which take place in the relationships between the bronchial and pulmonary vascular systems in a variety of conditions including emphysema and bronchiectasis. The studies of Liebow et al, (1949), Marchand et al, (1950) and Liebow (1953) indicate that broncho - pulmonary anastomoses exist normally and that in disease numerous abnormal vascular connections develop between the bronchial and pulmonary vascular systems. Such changes have been shown to have considerable functional significance in experimental animals but their importance in human disease remains to be determined.

In the field of clinical medicine, with the increasing use of the electrocardiograph and of radiological techniques, there occurred a rapid increase in knowledge of both circulatory and respiratory disease. A better understanding of the dynamics of both central and peripheral circulatory failure facilitated a precise and rational approach to the ultimate problems of treatment. In this phase of development, however, the pulmonary circulation remained relatively inaccessible.

The study of respiratory diseases followed similar lines and stayed, in the main, divorced from cardiology. The old concept of "emphysema heart" remained, but it is surprising how little attention was paid to cardiac failure in the course of other chronic respiratory diseases. Perhaps the difficulty in dissociating diseases such as bronchiectasis and pulmonary fibrosis from emphysema itself played a part in the relatively slow growth of the broader concept of pulmonary heart disease.

It is significant that, during this period spirometric techniques were being used by numerous workers in the study of respiratory function in health and disease. Much valuable information was obtained. But it is fair to say that difficulty in interpretation of the rather varied results, which might be obtained in similar pathological states, prevented the general adoption of such methods.

Physiologists, in the meantime, had been actively investigating the pulmonary and bronchial circulations by the employment of animal experiments. The techniques, which involved the study of virtually everything from excised portions of pulmonary vessels to nearly intact animals, were dictated by circumstances and the need to control or eliminate the numerous variables which influence the pulmonary blood flow in the intact animal. These variables include differences in cardiac output, variations in intra - thoracic and intra-pulmonary pressure and the incidental effects of nervous or pharmacological stimuli on the heart.

The literature on this subject is well reviewed by Wiggers (1921) and Daly (1933). In summary, it appeared that pulmonary blood flow was largely governed by the output of the right ventricle. In view of the fact that extensive vascular blockage or destruction was required before there was an increase in pulmonary blood pressure, it seemed that the pulmonary vascular bed had a great potential functional reserve. Initially there was little evidence of active vasometer control and it was thought that the lung vessels passively accommodated themselves to variations in cardiac output. However, more refined observations showed that the pulmonary vessels did in fact respond, by constriction, to adrenalin and to sympathetic stimulation. This response was much less dramatic than that in the systemic circulation. It appeared that, in normal circumstances, the bronchial circulation contributed but little to the total pulmonary blood flow. However, there were indications that, if the normal pressure relationships were disturbed, the amount of blood coming from the bronchial arteries could increase considerably.

More Recent Developments

By the pre-war period a considerable body of knowledge had accumulated but the means of applying this to clinical medicine had still to be discovered.

However, almost unnoticed, Forssmann (1929) passed a ureteric catheter into his own right ventricle through an incision in his left ante-cubital vein. Unfortunately it was some time before Forssmann's achievement was exploited and it was not until Cournand and Ranges (1941) published their account of catheterization of the right atrium that the method of cardiac catheterization brought the right heart and the arterial side of the pulmonary circulation under direct observation. Since then numerous publications, including those of Cournand and his co-workers, have established the value of catheterization both as a diagnostic and as a research procedure.

In the same period the introduction of cardioangiography by Castellanos et al, (1937) provided a means of visualizing anatomic changes in pulmonary circulation.

Following the first successful ligation of a patent ductus arteriosus by Gross and Hubbard (1939) and the work of Blalock and Taussig (1945) on the surgical treatment of congenital pulmonary stenosis, cardiac catheterization became intimately linked with cardioangiography in the field of cardiac surgery. With the increasing need for information of pressures in the right atrium, especially in relation to mitral disease, attempts at left sided catheterization were made. But, according to Friedberg (1956), the method which is likely to be adopted in the future is direct puncture of the left auricle through the posterior chest wall.

Meanwhile respiratory physiologists were developing new methods of studying pulmonary function. Many of these techniques are, in their performance, independent of cardiac catheterization and give information concerning lung volumes or external respiration. However, those concerned with the diffusion of gases from the lungs to the blood, in part at least, reflect the activity of the pulmonary circulation, and the data acquired from catheterization studies has been invaluable in the analysis of the significance of such tests.

Detailed accounts of the theory and practical application of these tests and their relationship to pulmonary blood flow are given in the excellent review by Lilienthal and Riley (1954) and by various authors in the monograph on pulmonary emphysema edited by Barach and Bickerman (1956).

Modern Applications

Thus, in recent years both clinician and physiologist have developed techniques and made measurements which have increased our understanding of the behaviour of the pulmonary circulation in health and in disease—sound methods of estimating pulmonary function have clarified concepts of the diffusion of gases in the lungs and the role of the pulmonary circulation in this function. Fortunately, at the same time, the fundamental studies of Burton (1953) on hemodynamics in blood vessels have provided more precise measurements of vascular performance. Out of these complementary advances has come the realization that separation of respiratory from circulatory function is artificial and misleading: "indeed, the interweaving of these two functions cannot be unravelled under normal conditions, and they become inextricably linked in disease." Thus, diagnostic and research units devoted to problems and diseases of the heart or lungs, have tended to become "cardiorespiratory" in scope rather than merely devoted to one or other of the two systems.

This thesis of the unity of the two systems can be best illustrated by a consideration of the effect of heart disease on the lungs and the effect of lung disease on the heart. The latter is now well known and has the distinction of a name of its own (cor pulmonale), whereas the former is perhaps less familiar particularly as it affects the pulmonary circulation.

The Effect of Heart Disease on the Lung

Prominent effects on pulmonary circulation occur in mitral stenosis, left ventricular failure and congestive heart failure, and in some congenital heart conditions.

Mitral Stenosis. Normally blood flow through the mitral orifice results from the excess of left atrial over left ventricular pressure in diastole. This diastolic atrioventricular pressure difference is termed the mitral gradient and is normally of the order of 1 mm. of Hg. or less. But when the cusps of the mitral valve are fused and rigid there is a mechanical obstruction to flow which results in an increased left atrial pressure; it varies from 10 to 40 mm. of Hg. so that the gradient varies between 5 and 30 mm. of Hg. depending on the severity of the stenosis. As there are no valves between the left atrium and the pulmonary veins, the elevated left atrial pressure is transmitted to the pulmonary veins and capillaries. Since the right ventricle maintains its normal output despite this increased resistance (by more forceful contraction in accordance with Starling's law), the pressure rises in the pulmonary artery and in the right ventricle. Prolonged pulmonary hypertension thus produced eventually results in hypertrophy of the right ventricle and in sclerosis and narrowing of the pulmonary arterioles. The pulmonary vascular changes thus induced may constitute as important an obstacle to flow as the original mitral obstruction. This barrier may serve to protect the congested capillaries from dangerous rises in pressure on exertion and thereby reduce the likelihood of episodes of acute pulmonary edema—but at the cost of ultimate right ventricular failure.

But what is happening in the pulmonary parenchyma while these vascular changes are occurring? The congested capillaries become widened, elongated and beaded, and project into the alveolar lumina, and the arteriolar walls become thickened. The alveolar walls also become thickened from interstitial edema, increased collagen and widening of the capillary basement membrane. These changes interfere with pulmonary ventilation and with gaseous exchange so that pulmonary function studies show reductions in vital capacity, maximal breathing capacity, inspiratory capacity and expiratory reserve, and sometimes reduced arterial oxygen saturation. The findings in these tests correlate well with the degree of clinical disability.

Provided pulmonary congestion has not been present for too long, these changes are reversible to a considerable degree. Successful mitral valvotomy results in a marked fall in left atrial pressure, and a reduction in the left atrioventricular gradient. There is then a gradual but variable reduction in pulmonary artery pressure and improvement in pulmonary function tests. However, if severe congestion has been present for a long time, pulmonary vascular changes become irreversible and removal of the obstruction at the mitral valve fails to improve the patient. It is apparent

that proper timing of surgical intervention is necessary to ensure maximum benefit from the operation.

Left Ventricular Failure and Congestive Heart Failure. The commonest cardiac diseases mainly damage the left ventricle so that failure of the left side of the heart is the most frequent form of congestive failure — though ultimately failure of one side places a strain on the other with resulting generalized cardiac failure. The consequence is pulmonary engorgement, which is the dominant pathological finding and is the basis of the dominating symptom of dyspnea in varying degrees of severity. Grossly, such lungs are larger, more turgid and less resilient than normal. Microscopically they show capillary congestion and thickening of alveolar septa by interstitial edema. This pulmonary congestion disturbs respiration by interfering with diffusion of oxygen but more importantly by producing a rigidity in the lungs which restrains inspiration and expiration. Thereby results the disordered breathing (dyspnea) characteristic of cardiac failure.

The shallower, more rapid respiration of congestive heart failure is associated with a reduced vital capacity and a reduced maximal breathing capacity; these and other quantitative measurements are merely indices of pulmonary rigidity.

It is thus apparent that much of the functional disturbance in the commonest forms of heart failure lies in the congestive effect of heart failure on the lungs. Likewise it is clear that the success of therapeutic measures will depend on their ability to relieve the offending pulmonary congestion.

Congenital Heart Disease. Little attention was paid to the pulmonary circulation in congenital heart disease until operations capable of increasing pulmonary blood flow began to be done on cyanotic patients. It rapidly became apparent that some cyanotic individuals on whom an anastomotic operation was to be done already had a severe pulmonary hypertension, a condition which would not allow an increased pulmonary flow were the procedure performed. As cardiac catheterization data accumulated in patients with some anatomic defect, it was soon apparent that pulmonary artery pressures varied from normal to very high; physiological and clinical behavior in these patients was so diverse that superficially it was hard to believe that they were all affected by the same cardiac malformation. It has now been realized that this variability is dependent on the pulmonary vascular response. This realization has been recently clarified by Dr. Jesse E. Edwards (1957) in the 1956 Lewis A. Conner Memorial Lecture: "Functional Pathology of the Pulmonary Vascular Tree in Congenital Cardiac Disease."

Through a very thorough analysis of an extensive experience, Edwards demonstrates that in many varieties of congenital heart disease the responses of the pulmonary vessels are of paramount importance in regulating the circulation. In some instances the pulmonary vascular responses are an integral part of the cardiac malformation; in others they are complications. With varying pulmonary vascular response the same anatomic defect is associated with different functional manifestations. Good examples of this are seen in Eisenmenger's complex and in the Tetralogy of Fallot where the clinical and hemodynamic effects of the anatomic malformations are so variable that it seems desirable to abandon these names in favor of a number of functionally descriptive terms as "ventricular septal defect with mild pulmonary stenosis and left-to-right shunt." Edwards' important paper very convincingly demonstrates the interdependence of the heart and pulmonary circulation in determining the ultimate outcome in congenital heart disease.

The Effect of Lung Disease on the Heart (Cor Pulmonale)

Cor pulmonale has been defined by Richards and Fishman (1956) as a heart which has a significant degree of dilatation, hypertrophy or failure, secondary to disease of the lungs. It is usually chronic, but may be subacute or even acute. According to Wood (1956) clinical recognition has become much more frequent in recent years and it now constitutes about 5 to 10 percent of all cases of organic heart disease.

The fundamental causative mechanism of cor pulmonale is an increase in the pulmonary vascular resistance and the consequent increase in pulmonary blood pressure. These changes, in turn, have been attributed to a variety of mechanisms (Friedberg, 1956): 1) anatomic reduction of the pulmonary vascular bed: 2) anoxia, which may act by a) pulmonary vasoconstriction, b) increased pulmonary blood flow, or c) increased blood viscosity; 3) increased intra-alveolar pressure; 4) increased bronchomotor tone; 5) bronchial-pulmonary shunts; and 6) secondary pulmonary arteriosclerosis. Of these factors, the most important is anatomic reduction of the pulmonary bed; anoxia is the next most important factor. Significance of the other factors is not well established. It is of interest that broncho-pulmonary shunts have been established in chronic pulmonary disease and it is possible that they play a role in the development of pulmonary hypertension.

The commonest cause of Cor pulmonale is emphysema where alveoli are distended and thereby result in obliteration of capillaries. This eventually leads to increased pulmonary vascular resistance and pulmonary hypertension. The alveolar abnormality also reduces the capacity of the lung to oxygenate and accordingly an element of hypoxia is added. It is this hypoxia which stimulates the increased cardiac output often found in Cor pulmonale. It is obvious that the necessity of maintaining a high output, and this against an increased pulmonary vascular resistance, greatly increases the work of the right ventricle. As in

the case of systemic hypertension, the ventricle responds by hypertrophy, but compensatory mechanisms may eventually become inadequate and failure then occurs.

It follows from the complexity of the factors involved in the pathogenesis of Cor pulmonale, that it is often difficult or impossible, on clinical evidence, to determine which is the most important. With the wider use of the methods now available for investigation of the circulatory and respiratory disturbances involved, it is possible that more precise identification of causative factors, particularly those that are reversible, will lead to more effective and less empirical treatment.

Summary and Conclusion

An attempt has been made to outline knowledge of the pulmonary circulation from earliest times. It is apparent that awareness of the significance of the pulmonary circulation was delayed long after the systemic circulation was comparatively well understood. The reason for this was, in part, the anatomical inaccessibility of the pulmonary circulation, and in part, its complex relationship to cardiac and pulmonary function. In recent years the development of new methods of studying cardiac and pulmonary circulatory dynamics has provided direct means of investigating cardiopulmonary vascular physiology. These methods are finding increasing application in clinical medicine where, in conjunction with improved tests of respiratory function, they have resulted in a fuller understanding of the integrated nature of cardiorespiratory disease.

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Cardiac Catheterization in Evaluating Patients With Mitral Stenosis for Commissurotomy

David H. Stein, M.D., F.R.C.P. (C)

The rapid advances made by the cardiac surgeons in the past decade have created new problems for internists and cardiologists. Diagnoses hitherto made with relative ease have become much more difficult with the additional burden of determining operability.

The appreciable risk attached to most cardiac surgery requires that precise anatomic and physiologic diagnoses be made in order to ensure operating only on patients likely to be helped by the procedure. While this is more applicable to congenital heart lesions, it is no less important in acquired heart disease such as mitral stenosis.

Patients with isolated or "pure" mitral stenosis are relatively easy to diagnose and evaluate as surgical candidates. Unfortunately, a large number of patients with mitral stenosis have associated valvular lesions such as mitral or aortic incompetence or murmurs suggestive of one of these lesions. Selection of such patients for mitral commissurotomy may be very difficult. The presence of an apical systolic murmur is often very vexing as such murmurs have been noted in "pure" mitral stenosis as well as when mitral incompetency has been proven present at operation.

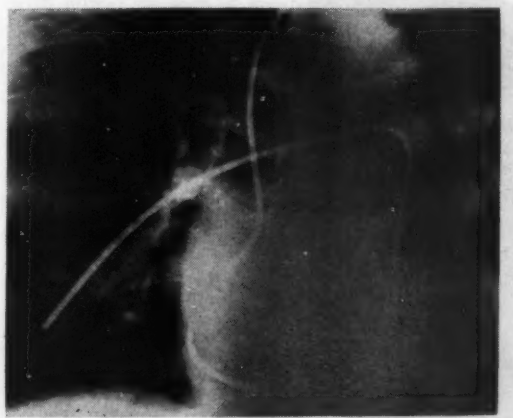
Careful clinical examination as well as radiologic and electrocardiographic studies will usually prove adequate in culling out most of these problem patients who do have predominant mitral stenosis. There will remain a small group of patients in whom additional studies must be performed in order to arrive at a decision regarding operability. Hemodynamic information obtained from cardiac catheterization will usually enable such a decision to be made. It should be stressed at this point that the associated presence of other valvular deformities or murmurs suggestive of them, does not contra-indicate mitral commissurotomy providing the mitral stenosis is the predominant lesion. It is the object of this paper to review some of the hemodynamic findings obtained by cardiac catheterization on patients with mitral stenosis and/or insufficiency and to outline the indications for these procedures.

First, it is appropriate at this point to draw attention to the contributions of two men, each of whom, in his time, established a new scientific basis for hemodynamic studies of the circulation. It is fitting that while we are commemorating the three hundredth anniversary of William Harvey's death, the medical world is presently paying tribute to a contemporary, Dr. Werner Forssman, who was recently awarded a Nobel prize in Medicine for the introduction of cardiac catheterization. This technique, which has made possible the study of intracardiac events in living humans, has been widely adopted as a research tool as well as a diagnostic aid. Much valuable information has already been accumulated regarding hemodynamics in normal and diseased states.

Probably thousands of patients with mitral stenosis of varying degrees have been studied by right heart catheterization in various medical centres. The information thus obtained allows an objective assessment of the severity and grade of the mitral stenosis as well as the associated pulmonary vascular changes.

The main hemodynamic measurements obtained from right heart catheterization, are, the cardiac output, pulmonary artery and pulmonary "wedge" pressures. These parameters are recorded during exercise as well as at rest. The "wedge" pressure, obtained by forcing the catheter into the most distal branch possible of one of the pulmonary arteries, has been found to correlate quite accurately with the level of left atrial pressure[1]. Figure 1 shows the cardiac catheter in the wedge position.

Figure 1



The following table outlines representative hemodynamic values obtained in various patients with uncomplicated mitral stenosis as proven at surgery.

Representative Hemodynamic Values Obtained by Right Catheterization in Patients with Uncomplicated Mitral Stenosis:

	Cardiac Output Litres/Min.		Pulm. Artery Pressure (mm Hg)		Pulm. Wedge Pressure (mm Hg)	
	R	EX	R	EX	R	EX
Group (1)	3 - 4	6 - 8	25/10	50/30	10-15	25-30
Group (2)	2 - 3	4 - 6	75/45	90/60	20-25	25-30
Group (3)	2 - 3	4 - 6	100/80	120/85	25-30	25-30
Normal	4 - 6	10-15	25/10	25/10	8-12	8-12

R — Resting State
EX — During Exercise

These patients are arbitrarily divided into three groups: (1) those with symptoms of pulmonary congestion only on moderate or severe exertion, (2) those with symptoms of pulmonary congestion on mild exertion or at rest, (3) those who have developed frank right heart failure with edema, hepatomegaly and engorged neck veins.

Since most of the surgical candidates are found in groups (1) and (2), it is of interest to correlate these catheterization findings with the pathologic physiology as well as the clinical picture. The less severe degrees of mitral stenosis as in group (1) have a relatively normal cardiac output as well as pulmonary artery and wedge pressures at rest. However, on exertion, the mechanical impedance to blood flow at the stenotic mitral valve causes the pressure to rise in the left atrium. This pressure rise is transmitted back to the pulmonary veins and pulmonary capillaries. When the pressure exceeds thirty mm. of mercury, pulmonary edema ensues. This pressure rise with slight increase in blood flow is the factor limiting the response of the cardiac output to exercise.

Patients in group (2) have more severe mitral stenosis resulting in elevated pressures in the resting state with a very limited rise in cardiac output possible on exercise. Many of these patients have pulmonary capillary pressures bordering on the edema level even when resting.

Those in group (3) have developed right ventricular failure as a consequence of the pulmonary hypertension due to the chronic pulmonary congestion and the hypertrophic and obliterative changes which take place in the smaller pulmonary arteries and arterioles.

Right heart catheterization is of little help in establishing the diagnosis of mitral stenosis, but does provide an accurate means of estimating the severity of the lesion. This has proven helpful in evaluating patients whose histories are not reliable for one reason or another. Having determined the cardiac output and pulmonary artery and wedge pressures, one can calculate from Gorlin's hydraulic formulae[2] the area of the mitral valve orifice as well as the resistance offered to blood flow by the vascular changes in the lungs. Also, signs and symptoms may be correlated with pulmonary vascular resistance, pulmonary hypertension and cardiac output.

Catheterization studies on patients found to have predominant mitral regurgitation at surgery, have shown somewhat lower pulmonary artery and wedge pressures than patients with pure mitral stenosis. Also the cardiac outputs tended to be

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higher but the differences are not sufficient to distinguish between mitral stenosis and regurgitation as the predominant lesion when both are present[3].

Right heart catheterization has proven useful in the evaluation of patients following mitral commissurotomy. If subjective improvement of the patient is the sole yardstick applied, the evaluation of this operation has been found to be extremely difficult. Many patients reportedly much improved following commissurotomy have been found to have unaltered hemodynamic values. Sudden relapse and rapid deterioration is not uncommon in such patients.

A fairly recent development in the physiologic approach to the investigation of cardiac disease has been the introduction of left heart catheterization via a needle passed into the left atrium either through a bronchoscope[4] or more commonly through the posterior chest wall[5]. While this procedure may carry a higher morbidity in terms of pneumothorax, hemopericardium, hemoptysis and other pulmonary complications, it allows precise diagnosis of mitral and aortic valve deformities. A thin catheter is guided through the needle into the left atrium and thence into the left ventricle. Pressure measurements made through this catheter then will demonstrate the pathognomonic pressure gradient across the mitral valve in diastole, which is diagnostic of mitral stenosis. The pressure gradients measured across stenotic mitral valves have been found to range from five to twenty-five mm. of mercury. Varying with the blood flow across the mitral valve at the time of the measurements, the higher pressure gradients between the left atrium and ventricle were generally associated with mitral stenosis of greater severity.

In addition, the pressure pattern obtained in the left atrium may reveal a regurgitant wave during ventricular systole indicative of mitral regurgitation.

Very complete and informative data has been obtained to date by simultaneous right and left heart catheterization. However, it should be emphasized that no one suggests that such procedures be carried out on all prospective candidates for mitral valve surgery, or even on all such patients with atypical or complicated features. A small number of these patients with loud apical systolic murmurs or unusual clinical, electrocardiographic or radiologic findings may require right and/or left heart catheterization before a definitive diagnosis can be made. As more of these studies are performed our knowledge and understanding of the hemodynamic changes in these valvular deformities will be advanced. Consequently, we will become better clinicians which will definitely decrease our reliance on these physiologic studies to provide accurate diagnoses.

Summary and Conclusions

Generally speaking, patients with predominant mitral stenosis will present with clinical, radiologic and electrocardiographic evidence of right ventricular overload regardless of the presence of associated valvular lesions or murmurs suggestive of them.

Right heart catheterization is not helpful in diagnosing mitral stenosis or determining if the mitral stenosis is the predominant lesion. However, it provides objective measurement of the severity of the disorder and is helpful in objectively assessing the benefits of mitral valve surgery.

Left heart catheterization does provide precise diagnoses of mitral stenosis and/or regurgitation.

As our understanding of these disorders is enhanced by the data accumulating from heart catheterizations, our need for such procedures to aid us in selecting patients for mitral valve surgery will diminish.

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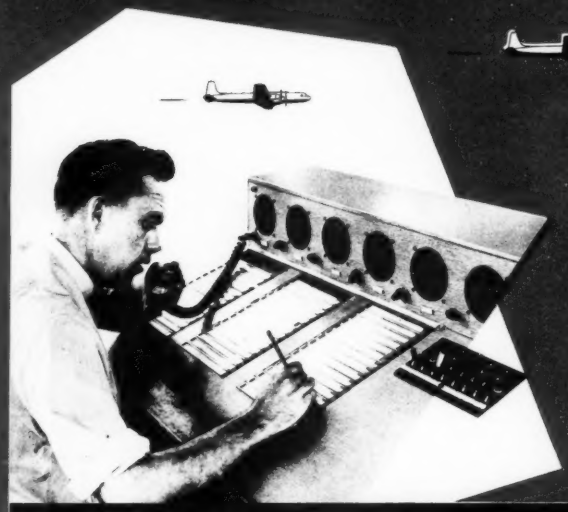

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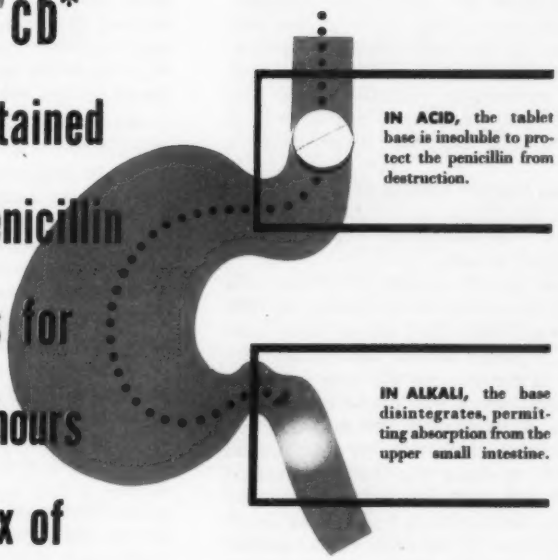

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Patent Ductus Arteriosus*

Harold W. Chestnut, M.D.**
and
Colin C. Ferguson, M.D.*

Just over three hundred years ago in the year 1628, William Harvey, a physician in London, published his treatise entitled "Anatomical Study on the Motion of the Heart and Blood"[1].

It is a far cry from that celebrated discovery to the rapid strides now being made in the correction of defects in the cardiovascular system. However, surely no little credit is due to the man who made this momentous contribution to the science of medicine and who started the volume of study and research which has made these advances possible.

Patent Ductus Arteriosus

Patent ductus arteriosus is, of all congenital anomalies of the cardiovascular system, most amenable to surgical attack.

Ligation of the patent ductus arteriosus was first suggested by John Munro[2] of Boston in 1907 but it was not until 1938 that Gross[3] first successfully ligated a patent ductus arteriosus. By so doing, he opened the way for the ever broadening field of cardiac surgery. While Gross first ligated the ductus in continuity, he soon realized that there were certain limitations to this method in that in only 80% of cases was there permanent complete closure[4]. In 1940 Touroff[5] divided and closed a patent ductus arteriosus, and in 1944 Gross[6] reported his technique for division of the ductus which he now does in all cases. Due to these advances, patients with patent ductus arteriosus are being operated in centres throughout the world with excellent results and an extremely low mortality rate. (Gross[7] reports a mortality less than 0.5% in uncomplicated cases.)

Etiology

During foetal life the blood is normally shunted away from the pulmonary to the systemic circulation via the ductus arteriosus which leads from the bifurcation of the pulmonary artery to the aorta. The ductus remains patent until shortly after birth when it becomes obliterated, due to diminution and finally cessation of blood flow through it as the pressure in the pulmonary artery becomes equal and subsequently falls below that of the aorta.

If obliteration does not occur, blood is shunted from the aorta to the pulmonary artery. The amount of blood thus diverted varies according to the size of the ductus, but may amount to as much as 70% - 90% of the blood coming from the left ventricle. It is not known why obliteration of the ductus fails to occur, but it is interesting to note that German measles affecting the mother in the first trimester of her pregnancy may result in an infant possessing a patent ductus often associated with other anomalies such as congenital cataract and cleft palate.

Symptoms and Complications

Typically children with a patent ductus arteriosus are asymptomatic and appear quite healthy with normal activity and endurance. As they become older they may begin to tire on exertion. If the ductus is small and if infection does not supervene, life may be lived out to its normal duration. Yet this is not often the case, and two thirds of patients with an open ductus develop either failure or subacute bacterial endarteritis before the age of forty.

In a significant number of cases, there is retardation of growth which may be quite striking.

It is important to remember that a patent ductus arteriosus may produce atypical signs and symptoms in infancy. It occasionally may be the only cardiac lesion responsible for cardiac failure, and the importance of diagnosing the condition in these cases is stressed.

The complications of patent ductus arteriosus may thus be listed as follows:

1. There may be retardation of growth, diminished exercise tolerance and recurrent pulmonary infections.
2. With increased cardiac output, the heart is overworked and eventually left heart failure may result.
3. With continued increased pulmonary blood flows, pulmonary vascular changes may occur producing pulmonary hypertension, reversal of ductal flow, cyanosis, and eventually right heart failure.
4. Subacute bacterial endarteritis may occur.
5. In infancy, a ductus may be the sole cardiac lesion responsible for failure[8].
6. Rarely a ductus may undergo aneurysmal dilatation or rupture.

Diagnosis

Patent ductus arteriosus is more common in females than in males. Gross[6] reports a series of 611 cases in which 70% were females.

The condition is unaccompanied by cyanosis or clubbing in uncomplicated cases.

In the vast majority of cases the lesion can be diagnosed in a few minutes by physical examination. In these, a very characteristic murmur is present which is aptly described as machinery-like in character. The murmur is continuous, is accentuated in systole and is heard best over the second left interspace, along the sternal border. The murmur may replace the pulmonic sounds or the second pulmonic sound may be accentuated. In 50% of cases there is an accompanying thrill.

The heart is of normal or of only slightly increased size.

The systolic blood pressure is usually normal. The diastolic pressure is depressed according to the flow through the ductus. With increased pulse pressure, a bounding pulse may be present and

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there may be visible capillary pulsations in the nail beds.

Femoral pulsations are normal, a point in differentiating from coarctation of the aorta.

The chief value of the electrocardiograms is in differentiating patent ductus from other cardiac lesions. It may be normal or show slight left ventricular hypertrophy.

Cardiac fluoroscopy is of help in diagnosis and also in ruling out other cardiac lesions. The heart may be slightly enlarged and typically there is a prominence in the region of the pulmonary artery. Pulmonary vascular markings are increased and a hilar dance may in some cases be noted.

Diagnosis in Atypical Cases

In 95% of cases of patent ductus arteriosus the diagnosis can be made by the methods outlined above. In the remainder, however, resort will have to be made to special diagnostic studies which will confirm or deny the existence of an open ductus. These patients are usually infants with a systolic murmur only, or adults in some degree of failure.

In these individuals, with increased pulmonary engorgement, there is an increase in the pulmonary artery pressure. If this increases to the level of the aortic diastolic pressure, there will be no flow through the ductus during diastole and a systolic murmur only will be present. With a further increase in pulmonary artery pressure there may be no flow or even reversed flow. There then may be no murmur and the patient may be cyanosed.

To establish a diagnosis in these cases, an aortogram may be done by cutting down on the left brachial artery and injecting radiopaque material in a retrograde manner. In the presence of a patent ductus, simultaneous visualization of the aorta and the pulmonary artery will be obtained and on occasion the ductus itself may be visualized.

Cardiac catheterization may aid in positive diagnosis in atypical cases. By this means, it may be demonstrated that, as the catheter is pushed beyond the pulmonary valve, the blood is found to have an increased oxygen content, proving that arterial blood is entering the pulmonary artery presumably through a patent ductus arteriosus. Occasionally the operator may be fortunate enough to pass the catheter through the open ductus into the aorta.

Infants with Systolic Murmur Only

In the early weeks of life due to high pulmonary pressure and low aortic pressure there may be very little flow through a patent ductus and thus no murmur may be heard. In time the aortic pressure rises and the pulmonary artery pressure falls with an increasing flow through the ductus. With flow during systole only, a systolic murmur will be heard, and it may not be until the age of two or three years that the characteristic continuous murmur through systole and diastole becomes present.

In infants with a systolic murmur only, and with no symptoms, absolute diagnosis may not be necessary at this time. It is necessary, however, to examine the child at subsequent intervals at which the characteristic murmur may be heard.

However, in some infants there may be marked retardation of growth and cardiac failure. In these, it is important that early diagnosis be made so that life saving surgical intervention may be forthcoming.

Physical and laboratory examination may be useful in ruling out other congenital anomalies. However, often a definite diagnosis cannot be made, and in these instances a retrograde aortogram will confirm the diagnosis.

Results of Surgery

A total of 63 patients with patent ductus have been operated upon by one of the authors (C.C.F.). Thirty-four of these were done in Boston, and 29 in Winnipeg in the last three years. There was one fatality in the total series. This occurred in a premature baby weighing four pounds, who had been in severe cardiac failure since birth. The ductus was closed successfully, but at the conclusion of the operation there still remained a prominent thrill over the anterior surface of the heart. At autopsy, a large interatrial septal defect was found. One patient at thoracotomy was considered inoperable but has survived.

Of the 29 patients operated upon in Winnipeg, 22 (77%) were females, and seven were males. The average age at the time of operation was 8.8 years. (7 weeks—40 years).

In 19 of these patients the murmur was discovered on school health examination or was heard by the family physician during examination for some other illness. In 12 patients there was definite retardation of growth. In most, the diagnosis was relatively straightforward — the characteristic murmur being present in 25 cases.

There were no complications during operation except in one patient in which there was serious bleeding from the aorta which was eventually controlled. The average diameter of the ductus was 7 mm. The ductus was divided and the ends oversewn in 22 cases and ligated in 6 and not operated upon in one.

Postoperative complications were minor in all cases, there being no serious problems, and, except for the one death in the series, all made an uneventful recovery. One case developed a stitch abscess postoperatively — the only wound infection. The average postoperative stay in hospital including the day of operation was 12 days. (9-19 days).

Follow-ups have been done on all patients, though obviously the period of observation has been short. One patient, aged 40 years, five months later still had an enlarged heart, though it was

smaller than before operation. Pulsations, thrill and murmur had disappeared. Blood pressure had dropped from 185/60 to 160/100 and he was feeling well.

All others are well, free of symptoms, and have resumed normal activity. The characteristic murmur and thrill disappeared in all, although three patients have residual systolic murmurs. Blood pressures have returned to normal, and the heart sizes have diminished, as have the pulmonary vascular markings.

Illustrative Case Histories

1. E. R., female, age 10 years. This little girl was noticed to have a heart murmur for several years. Five months before her operation she developed an upper respiratory infection, followed by an episode of chills and fever. Her fever persisted, and two months prior to operation blood culture was positive for streptococcus viridans. She was diagnosed as having a patent ductus arteriosus with subacute bacterial endarteritis and was treated with antibiotics. The blood culture became negative. Examination revealed a pale, slight girl, small for her age. There was a loud machinery-like murmur heard best in the second left interspace and there was a palpable thrill over the pulmonary area; B.P. was 110/50. Chest x-ray was considered normal. At operation, the peri-ductus area was very vascular presumably because of the previous infection. The ductus was short with broad bases and measured 5 mm. at its narrowest point. The ductus was divided and the ends oversewn. Two years later she had remained well, and on examination there was no residual murmur or thrill and the chest was clear.

Comment

Patient with a patent ductus and superimposed subacute bacterial endarteritis. The infection, in this case, cleared on antibiotic therapy, but in two other patients the infection did not subside until after closure of the ductus.

2. Baby F., female, age 7 weeks. This baby was born two weeks prematurely with a birth weight of four pounds and had gained little since birth. She had had some respiratory difficulty at birth and had been intermittently in and out of cardiac failure. Examination revealed a very small pale listless baby. The heart was markedly enlarged. A loud systolic murmur was heard along the sternal border in the left second and third interspaces. The liver was enlarged. Peripheral pulses were prominent. Chest x-ray showed an enlarged heart with a prominent pulmonary artery and increased vascular markings. A retrograde aortogram was done which revealed the presence of a patent ductus arteriosus. Electrocardiogram was "normal." At operation a thrill was felt in the region of the pulmonary artery. Three ties were placed around a medium sized ductus. The thrill was diminished but still present. The systolic murmur remained but was diminished in intensity. The baby died one and a half hours postoperatively. Autopsy showed the patent ductus to be completely closed, but there was an associated very large secundem type interatrial septal defect.

Comment

This comprises the only fatality in the series. Death was due to persistent cardiac failure as a result of a residual undiagnosed cardiac abnormality.

3. K. W., male, age 3 years. This little boy had considerable retardation of growth and at the age of 3½ years weighed 24 pounds. His mother had had German measles early in her pregnancy and the patient was noted to have a congenital left cataract at birth. In addition to the lesion of the left eye, physical examination revealed a loud continuous machinery-like murmur, heard best in the second left interspace. A palpable thrill was present. B.P. was 110/50. Chest x-ray showed slight cardiac enlargement with a slight prominence of the pulmonary artery. Electrocardiogram was normal. At operation there was a prominent continuous thrill over the pulmonary artery. A moderately long ductus was present, which was broader at the aortic than at the pulmonary end. The ductus was divided and the ends oversewn. Postoperative course was uneventful and he was discharged 12 days postoperatively. There was no residual thrill or murmur.

Comment

Typical case of patent ductus with a history of German measles in the mother during early pregnancy.

4. L. P., female, age 4 months. This baby had had considerable difficulty since birth, being repeatedly in and out of cardiac failure. On examination a loud systolic murmur was heard over the entire precordium. The heart was markedly enlarged. Chest x-ray showed marked pulmonary vascular congestion. A retrograde aortogram was done which revealed a large patent ductus. A prominent thrill was palpable in the region of the pulmonary artery. A large ductus was present, which was only slightly smaller than either the aorta or pulmonary artery. The patent ductus was closed by two silk ties and in addition, a tie of umbilical tape. There was no residual thrill. The baby was discharged 15 days postoperatively after a satisfactory postoperative course. Three months later she was doing well. Chest x-ray showed the heart to be still enlarged though considerably diminished in size, and there was marked diminution of pulmonary vascular markings. A soft residual systolic murmur remained.

Comment

Atypical case with systolic murmur only, which was diagnosed by means of a retrograde aortogram.

5. S. Y., female, age 4½ years. This little girl had always been undersized, tired easily and had shortness of breath on exertion. Examination revealed a small, pale, sick looking child. A loud

grade 3 systolic murmur was heard in the mid-sternal region at the level of the 3rd interspace. The second pulmonic sound was accentuated. B.P. was 116/60. Chest x-ray showed considerable cardiac enlargement with a prominent pulmonary artery and markedly engorged lung fields. Cardiac catheterization was done which showed increased right ventricular and pulmonary artery pressure, and the catheter was seen to enter the aorta from the pulmonary artery. An extremely large ductus was present measuring 2 cms. in diameter, which was larger than the pulmonary artery. The ductus was divided and the ends oversewn. There was no residual thrill. Postoperatively there was some cardiac irregularity which shortly subsided. She was discharged 14 days postoperatively. Two months postoperatively she had gained well and no longer looked like a sick child. There was no residual murmur. Heart size had diminished considerably.

Comment

This is another example of an atypical case, in which cardiac catheterization established the diagnosis.

6. L. L., female, age 10 years. At the age of four years, this patient was admitted to the Children's Hospital because of a fever which quickly responded to penicillin therapy. A heart murmur was noted at that time, which by one observer was reported to be continuous and machinery-like in character. A chest x-ray was normal. One month later the child had a recurrence of her fever, accompanied by hoarseness and persistent cough. Chest x-ray at this time, revealed a large mass in the region of the pulmonary artery. A diagnosis of subacute bacterial endocarditis was made, and again the child responded to antibiotic therapy. Over the intervening years, x-rays showed the mass to remain the same size, but its wall became calcified.

At 8 years of age, cardiac catheterization was performed in Toronto, and a diagnosis of pulmonary stenosis with poststenotic aneurysmal dilation was made.

In 1955, the child was completely re-investigated. Angiocardiography revealed no communication between the mass and the aorta or the pulmonary artery.

Operation revealed a large solid mass, three inches in diameter, situated between the pulmonary artery and the aorta. No thrill was felt over the mass. The lesion was considered inoperable. The final diagnosis was a calcified thrombosed aneurysm arising in a previously patent ductus.

Comment

This child, with a patent ductus, developed subacute endarteritis. An aneurysm subsequently formed which later became thrombosed. At thoracotomy, however, the aneurysm could not be resected.

Summary

A brief account of the history, treatment, symptoms, signs and methods of diagnosis of patent ductus arteriosus has been presented along with a discussion of complications that may arise, and of special methods of diagnosis in atypical cases. A review of 29 cases operated upon in Winnipeg together with illustrative case histories has pointed out the satisfactory results of operative treatment.

Conclusions

1. Patent ductus arteriosus is a congenital cardiac anomaly which can be cured surgically.
2. Patent ductus arteriosus may be readily diagnosed by straightforward methods in the vast majority of cases.
3. Patent ductus arteriosus carries with it the risk of grave complications and shortened life expectancy.
4. Results of operation are extremely satisfactory and the operation carries a low mortality rate.
5. The importance of patent ductus arteriosus as a cause of heart failure in infancy has been pointed out.

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The Contributions of Surgery to Cardiac Function

1. Separation of the Pulmonary and Systemic Circulations

L. L. Whytehead, M.D., F.R.C.S. (Eng.)

Introduction

The direct effects of operative surgery are purely mechanical but from them may ensue profound alterations in function. This is as true in diseases of the heart as elsewhere and in this field the contributions of surgery are largely the relief of mechanical hindrances to the circulation.

Incomplete Separation of the Pulmonary and Systemic Circulations

The majority of anomalies which allow mixing of partially and fully oxygenated blood are congenital and they may be listed as follows:

A. Patent Ductus Arteriosus.
B. Atrial Septal Defects.
C. Atrio-ventricularis Communis.
D. Ventricular Septal Defects.

E. Aneurysms of the Aortic Sinuses of Valsalva which have ruptured into the Right Atrium or Right Ventricle.

F. Pulmonary Arterio-venous Fistulae.

It will be realized that these conditions are essentially arterio-venous fistulae which result in shunts of blood between the pulmonary and systemic circulations. The ability of the heart to compensate for these handicaps to efficient function may prevent the appearance of symptoms for variable periods which depend largely on the size of the shunt. The mechanism of compensation is fundamentally similar in all these anomalies. One of the ventricles has to increase its output by the volume of blood which passes through the shunt and the resulting hypertrophy can be detected electrically.

Direction of Shunt

Except in pulmonary arterio-venous fistulae, these defects, when isolated and uncomplicated, always result in a left-to-right shunt; i.e., the shift of fully oxygenated blood into partially oxygenated. This is because the pressures in the left heart chambers are higher than in the right chambers which in turn is due to the pulmonary resistance being less than the systemic. Consequently there is no cyanosis.

However, associated anomalies or ensuing complications may elevate the pressures in the right chambers above those in the left chambers so that the shunt is from right to left and partially oxygenated blood is shifted into fully oxygenated blood. The oxygen content of peripheral arterial blood is thus reduced and if one-third or more of the haemoglobin is reduced, cyanosis is apparent. This hypoxaemia causes polycythaemia and clubbing of the digits. In this connection, the relevant associated anomaly is pulmonary stenosis and the important complication is pulmonary arteriolar sclerosis. The latter condition results from prolonged excessive pulmonary blood flow in left-to-right shunts and leads to pulmonary hypertension which eventually is reflected in right ventricular and right atrial hypertension. The direction of shunting is therefore reversed. Pulmonary stenosis is often associated with a ventricular septal defect and then constitutes the condition known as Fallot's Tetralogy. The other two facets of this condition, namely over-riding of the aorta and right ventricular hypertrophy, are less important results of the two primary anomalies. Pulmonary stenosis may also co-exist with an atrial septal defect which, in a good proportion of patients with both lesions, is a functional foramen ovale rather than a true defect. The stenosis is more often valvular when the defect is between the atria and more often in the infundibulum or outflow tract of the right ventricle when the defect is between the ventricles.

Special Aids to Cardiac Surgery

Surgery can now offer satisfactory operations to correct most of the forms of incomplete separation of the major and minor circulations. However, the operative treatment must be applied before the development of pulmonary arteriolar sclerosis and an associated pulmonary stenosis must be simultaneously corrected.

Most of these anomalous communications between the heart chambers can only be repaired under direct vision with the heart widely opened. This necessitates interruption of the blood flow through the heart, but under normal conditions the duration of this interruption must be limited to three minutes to avoid anoxic damage to the brain. The body metabolism may, however, be reduced by lowering the temperature and with levels of hypothermia between 28° and 30° C, the period of interruption may be prolonged safely to six to eight minutes (Bigelow)[1]. This time is sufficient only for the repair of simple atrial septal defects and at this degree of hypothermia the ventricles tolerate incision poorly. The repair of other defects requires the use of apparatus to assume the pumping action of the heart and the gaseous exchange of the lungs for longer time intervals, i.e., some type of pump-oxygenator or artificial heart-lung machine. Of these it need only be said that the oxygenators, which also allow the venous blood to give up carbon dioxide, are of two classes.

(i) Those in which the blood is exposed to oxygen in a film to avoid bubble formation (Gibbon)[2].

(ii) Those in which oxygen is deliberately bubbled through venous blood, the bubbles being subsequently separated by the use of Silicone Antifoam compounds in a settling tube which provides forward flow (DeWall)[3].

Although a Pump Oxygenator permits wide opening of any heart chamber for long periods of time, the heart continues to contract regularly, metabolises oxygen and carbohydrates and air may be driven into the coronary circulation causing embolism. Two contrasting methods of dealing with these problems have been used. One employs retrograde perfusion of the coronary system through a cannula inserted into the coronary sinus; a large proportion of the perfused blood is recovered from the left coronary ostium and is found to have given up some of its oxygen (Gott)[4]. This supplies at least some of the metabolic requirements of the heart and prevents entrance of air into the coronary ostia. The other method consists of elective cardiac arrest, induced by injecting potassium chloride or citrate into the coronary circulation (Melrose)[5]. The heart muscle is at rest which makes repair easier technically and its metabolism is greatly reduced; air cannot be driven into the coronary ostia. When blood is again allowed to flow through the coronary circulation, the potassium is washed out and the heart action restarts spontaneously or with massage, although calcium supplements may also be neces-

sary. These two techniques must be considered experimental.

Patent Ductus Arteriosus

This condition is well enough known not to require description. It was first closed surgically by Gross[6] and the operation is now practised universally since it requires no special aids.

Atrial Septal Defects

Two main types may be distinguished. The simpler type has an inferior rim of septal tissue and is called the ostium secundum type in reference to its embryological origin. In the second type the defect extends down to the upper margin of the ventricular septum, the ostium primum type.

Secundum defects were first repaired by closed techniques without opening the right atrium except to introduce a finger to guide the placement of sutures. Satisfactory repair can be effected by Bailey's technique of Atrio-septopexy[7] and by Sondergaard's technique of Circumclusion[8]. However, repair is more certain by open technique and for secundum defects, hypothermia is a very satisfactory method (Swan)[9]. Septum primum defects are not satisfactorily repaired by closed techniques and require an exposure in excess of six to eight minutes; they should therefore be repaired with the aid of pump oxygenators.

Atrio-ventricularis communis

This complicated defect may occur in a variety of forms but basically the defect involves the adjacent parts of the inter-atrial and inter-ventricular septa with or without defects of one or both atrio-ventricular valves. The elaborate repair needed can only be undertaken with the use of a pump oxygenator, (Cooley)[10].

Ventricular Septal Defects

These usually involve the membranous portion of the septum and are accordingly closely related to the septal cusp of the tricuspid valve and the cusps of the aortic valve. Repair necessitates a ventricular cardiotomy and so the use of a pump oxygenator is necessary (Lillehei)[11].

Aneurysms of the Aortic Sinuses of Valsalva

Most of these are congenital but some are syphilitic or mycotic. They usually rupture into the right atrium or right ventricle. At least one successful repair of such an aneurysm which had ruptured into the right ventricle has been achieved (Sawyers)[12].

Pulmonary Arteriovenous Fistula

These fistulae may be multiple. They are, in effect, right to left shunts because a portion of blood carried by the pulmonary artery passes back to the heart by the pulmonary veins without being exposed to oxygen in pulmonary capillaries. The patients accordingly are cyanosed, polycythaemic and have clubbing. The treatment is to excise the affected parts of the lungs.

One may summarize the results of treatment by saying that excellent correction of these defects can be obtained in all types. The mortality for closure of patent ductus and for excision of pulmonary arteriovenous fistulae is low but the use of hypothermia and more especially of pump oxygenators to repair the other defects is as yet associated with a considerable mortality.

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The Functional Innervation of the Blood Vessels of the Skin and Muscles of the Limbs

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It is generally accepted that the neurogenic pathway of central control of the blood vessels in the limbs is via the sympathetic nerves, but there has been continuing investigation of the functions of the fibres supplying the vessels of the various parts of the extremities for many years. The greatest effort has been expended in elucidating the influence of the sympathetic nerves on the vessels of the skin and muscle, and recent work has indicated that beliefs formerly held may need revision.

A great deal of work has been carried out in animals where freer methods of experimentation can be employed than on the human subject. However, the functional innervation of the vessels varies from one species to another. For example, vasodilator fibres have been found in the sympathetic nerves supplying the muscles of the dog and cat but not in those supplying the muscles of the rabbit. The function of the vasomotor nerves in the human limb must, therefore, be investigated in the human subject.

It may help in understanding the progress of our knowledge in these matters if the essentials of some of the methods of investigation applied to human subjects are discussed. One of the major problems was how to measure the rate of blood flow in the various tissues of the extremity, such as skin and muscle, separately, or, at least, to tell whether the rate of flow increased or decreased and whether the change was dependent upon a change in the neurogenic vasomotor tone in the tissue under study. The following are some of the

important methods used in studying changes in the rate of blood flow.

Some Methods of Investigating Blood Flow

Measurement of Skin Temperature

An increase in the temperature of the skin under appropriate circumstances indicates an increase in the rate of blood flow through the skin although the increase in temperature is not linearly related to the increase in rate of flow. The skin temperature has usually been measured by means of a thermocouple — a pair of thermojunctions (usually soldered junctions of fine copper and constantan wire) in series in an electrical circuit. The junctions develop an electromotive force which is dependent upon their temperature. When one junction is applied to the skin and the second kept at some reference temperature, the voltage developed in the completed circuit is linearly related, within the physiological range of temperature, to the difference in temperature between the two junctions. The temperature of one junction being known, the temperature of the other can be calculated or read from a suitably- calibrated galvanometer or potentiometer.

Measurement of Heat Elimination

A second method of ascertaining changes in the rate of blood flow through the skin is to measure the rate of heat elimination from the skin. When the rate of flow increases, the skin temperature rises and more heat is given up to the environment and, if the environment is water in a calorimeter, the rate of heat elimination can be measured. This principle is most suitably applied to the study of changes in the rate of blood flow in the skin of the digits or hand.

Plethysmography

This method of measuring the rate of blood flow has played an important part in the study of blood flow in the human limbs. It is the only technique which measures the rate of flow in absolute terms expressed as ml. of blood flow/100 ml. of tissue/minute. When the venous return of a part under study is prevented, the blood collects in previously slack veins and the rate of swelling of the part is equal to the rate of flow of blood through the arteries supplying it. The part under study is sealed into a plethysmograph. This is simply a rigid container with accessory equipment for measuring the rate of swelling of the part within it when a pneumatic cuff around the extremity just proximal to the plethysmograph is inflated to a pressure which does not hinder the arterial flow beneath the cuff but effectively closes the veins under it. The cuff is inflated for a few seconds only at a time since the veins become filled with blood, and the rate of swelling is no longer related to the inflow through the arteries.

Plethysmography can be applied to almost any part of the extremity such as the finger, hand or forearm. But it measures the rate of flow in the whole part included within the plethysmograph and does not separate the flow in one tissue from that in another. There are two major circulations in the limbs from the point of view of volume flow—skin blood flow and muscle blood flow. The blood flow through bone, tendon, and other tissues is relatively small except in Paget's disease of bone, when bone blood flow may be very large and important. In the past it has been considered that plethysmography of the hand measures blood flow which is mainly through skin and that plethysmography of the forearm measures largely blood flow through muscle. In the light of recent work this is only true in certain circumstances. Certain tricks have been employed in conjunction with plethysmography to try to decide in which tissues the change in flow it measures is occurring.

Calorimetric Probe

Changes in the rate of blood flow in muscle can be detected by means of a calorimetric probe or sound[1]. This instrument consists of a needle-like probe which is inserted into muscle. The probe carries two thermojunctions separated by a short distance and comprising a thermocouple. One of the junctions is supplied with a tiny electrical heating wire. In use the current through the heating wire is adjusted to maintain a constant temperature difference between the two junctions. When the rate of blood flow in the neighborhood of the junctions is increased, more current is required through the heating wire to maintain the difference between the junctions. This method is useful in detecting changes in rate of flow, their direction and relative magnitude during a single insertion of the probe and in the immediate vicinity of the probe. However, the relative magnitude of the changes may be different if the probe is reinserted in a different portion of the muscle. Nevertheless, with certain precautions it will apparently give an accurate qualitative picture of the blood flow changes occurring in muscle.

Clearance Rate of Radioactive Substances

The rate of clearance of radioactive sodium from muscle after a small quantity is injected into the muscle is used to obtain a qualitative picture of changes in the rate of blood flow at the site of injection[2]. This is a useful procedure but has many limitations. The rate of clearance is dependent upon many factors, only one of which is the rate of blood flow. It is said to indicate changes in nutritive blood flow only and not variations in flow through nearby A-V anastomoses or other vessels not supplying true capillaries. The rate of clearance may vary greatly when injection is made into a different site in the same muscle, but qualitative changes in the rate of nutritive blood flow through muscle should be reflected in the rate of clearance. This technique may be applied to skin as well as muscle.

Oxygen Saturation of Venous Blood

Another way of following the direction of changes in rate of flow in either skin or muscle is to sample venous blood leaving these tissues and

measure its oxygen saturation. A sample of venous blood from muscle is obtained by passing a small plastic catheter into a vein at the antecubital fossa and threading it peripherally past the venous valves so that it lies in a deep vein of the forearm which drains the muscle[3]. Intermittent or continuous samples of venous blood can be obtained. A similar catheter in a superficial vein of the forearm allows sampling of venous blood from the skin vessels. It is said that under the proper circumstances no significant mixing of the blood from skin and muscle occurs at this level. Changes in oxygen saturation of the blood will represent changes in the rate of muscle or skin blood flow provided the metabolism of the tissues remains unaltered.

With these methods in mind we can pass on to a consideration of the functional innervation of muscle and skin vessels.

Functional Innervation of Muscle Vessels

For some years it was believed that the muscle vessels were supplied with sympathetic vasoconstrictor nerves whose activity could be inhibited by heating the subject[4]. This belief was based on the evidence[5] that (a) anaesthetizing the motor nerves of the forearm caused an increased rate of blood flow in the forearm as measured by plethysmography; (b) a similar increase in forearm flow occurred if heat was applied to the body of a normal subject, but not in a subject whose arm was sympathectomized; (c) the increase in flow occurred even if iontophoresis of adrenaline into the forearm skin was carried out just before body heating to abolish blood flow in the skin. The result of the latter procedure indicated that the increase in flow occurred in the muscle vessels, since it was believed all circulation in the skin was abolished by the adrenaline and the flow in other tissues was too small to account for it. However, it has recently been found that more intensive iontophoresis of adrenaline into the skin will prevent the increase in forearm blood flow during body heating[6]. The increase in flow during body heating was, after all, occurring in the skin and not in the muscle vessels. This conclusion is supported by observations made with the calorimeter probe inserted into the muscles of the calf during body heating[7]. No increase of blood flow through the muscles was indicated by the instrument. It has also been shown that the oxygen saturation blood from the skin vessels is increased during body heating, but not that of blood from muscle veins[8].

The rate of clearance of radioactive sodium injected into the gastrocnemius muscle is not increased by body heating and, in fact, may be decreased[9]. Although this fact is supporting evidence, it could not be concluded from this alone that an increase in blood flow did not occur in the muscles since the clearance rate is thought to measure nutritive flow only. It is also known that the rate of clearance from muscle is not increased during intravenous adrenaline infusions when other techniques show that the total flow through muscle does increase[10]. Furthermore, this technique does not always indicate an increased blood flow in the skin during body heating[9]. The increased forearm flow during body heating will be considered again later.

Although it has been shown that body heating does not increase the rate of blood flow through muscle, the fact remains that anaesthetization of the motor nerves of the forearm causes an increase in forearm blood flow. That this increase is in muscle vessels as well as in skin vessels is indicated by the observations that oxygen saturation of blood from muscle veins is increased[11]. Furthermore, any increase in total blood flow in the forearm on blocking the cutaneous nerves could not account for the whole rise in flow when the motor nerves were blocked[5, 12]. The muscle vessels, therefore, seem to be endowed with vasoconstrictor nerve fibres.

The muscle vessels are believed to be provided with sympathetic vasodilator as well as vasoconstrictor fibres. This conclusion was based solely on evidence obtained during fainting following experimental hemorrhage[13, 14]. It was discovered that the blood flow in the forearm, measured by plethysmography, was increased during a faint in spite of a precipitous fall in blood pressure. The increase did not occur during fainting in sympathectomized subjects and was, therefore, of nervous origin. It was believed to occur in muscle because the rate of blood flow in the hand, where there is little muscle and a lot of skin, decreased during the faint and the skin became pale. It was possible the increase was due to inhibition of vasoconstrictor nerves. But it was concluded that activation of sympathetic vasodilator nerves was responsible because the maximum rate of blood flow through the forearms with all nerves intact during a faint was greater than the maximum rate through the forearms whose motor nerves had been anaesthetized to abolish neurogenic vasoconstrictor activity (and incidentally any vasodilator nerve activity). If the increased flow through the muscle was simply due to inhibition of vasoconstrictor nerves to muscle, the flow through the normal and nerve-blocked forearms should have been the same during the faint. The results suggested that the maximum rate of flow was about 60% greater in the intact forearm than in the nerve-blocked forearm, while the actual difference was of the order of 2 ml./100 ml. of forearm/min.

The interpretation of these results may need to be reconsidered if it is found that anaesthetization of the motor nerves of the forearm actually blocks the vasomotor nerves to all of the forearm skin as well as those to the muscle[15]. Furthermore, it has become evident from the work with the adrenaline iontophoresis mentioned above that a pale skin does not mean that an increased flow is not occurring in the deeper cutaneous tissues and, in fact, it is claimed that there is an increased heat elimination from the hand during emotional fainting[16]. It is possible that the functional innervation of

the skin of the hand is different from the skin of the rest of the arm so that what happens to the blood flow in the hand may not reflect the changes occurring in the forearm skin.

The ultimate function of vasoconstrictor or vasodilator nerves supplying the muscles of the limbs is not established. The vasodilator nerves were apparently activated during fainting but their normal function is unknown. Neither the constrictor nor dilator fibres seem to play any part in body temperature regulation. No evidence has been found that increased blood flow through the muscle brought about by alteration of the activity of the nerves enhances the effectiveness of the blood supply to exercising muscle in the normal human subject[4]. Other factors such as the local accumulation of metabolites are apparently quite capable of dilating at least the smaller muscle vessels and increasing the blood flow to levels much higher than those obtained by nerve block or sympathectomy[17].

It is known that passive changes in body posture cause reflex changes in forearm blood flow[18] and it has been found that passively elevating the legs in a supine subject will increase the rate of flow through the muscles of the forearm but not through the skin[19]. The increaased flow is dependent upon the sympathetic nerves and it is suggested that the nerves supplying the muscle vessels play a part in blood pressure regulation. It has not been decided whether vasoconstrictor or vasodilator fibres or both are involved.

Functional Innervation of Skin Vessels

It is well-known that the skin of the hands and feet is supplied with sympathetic vasoconstrictor nerves. Interruption of the sympathetic fibres supplying these areas by operation or anaaesthetization of the nerves leads to vasodilatation and a large rise in skin temperature. The fibres concerned are adrenergic and their influence may be abolished by adrenergic blocking agents. Body heating acting through the central nervous system inhibits these fibres and causes increased blood flow through the skin with increased heat elimination[20].

The skin of the arms proximal to the hands is probably also supplied with vasoconstrictor nerves, but anaesthesia of the cutaneous nerves of the forearm causes little or no rise in forearm skin temperature[21] in contrast to the great rise which may be observed in the skin of the hands when its nerve supply is blocked. Blocking the cutaneous nerves of the forearm produced only a slight increase, or none at all, in the total forearm blood flow as measured by the plethysmograph[5, 12]. However, vasodilatation in the skin on blocking the cutaneous nerves of the forearm is also indicated by an increase in oxygen saturation of the venous blood leaving the forearm skin vessels[15].

In considering the possibility of sympathetic vasodilator nerves supplying the vessels of the skin, it is necessary to think about the skin of the hands separately. The presence of such nerves in the skin of the hands has long been debated. Lewis and Pickering[20] thought that they existed because the vasoconstriction occurring in the fingers of patients with severe Raynaud's disease when they were in a cold environment could be released by warming the patient's body (the hands remaining in the cold) but not by anaesthetizing the nerves supplying the constricted fingers. They postulated that the body heating activated vasodilator nerves passing to the constricted skin vessels as well as inhibiting the activity of their vasoconstrictor nerves, whereas the nerve block simply inhibited the vasoconstrictor nerves. In normal subjects under the same circumstances the removal of vasoconstrictor influence by nerve block is sufficient to cause vasodilatation and a rise in skin temperature, but in fingers afflicted with Raynaud's disease some positive vasodilator influence seemed to be necessary to overcome the abnormal constrictor tendency of the vessels. The observations of Lewis and Pickering were confirmed[22], but no one has been successful in demonstrating the activity of vasodilator nerves in the hand or foot in normal subjects[23, 24, 25, 26] and possibly there is a different explanation for the observations in patients with Raynaud's disease from that postulated by Lewis and Pickering.

Although the existence of vasodilator nerves to the skin of the hands is in doubt, there is good evidence that such nerves supply the skin of the more proximal parts of the limb. Grant and Holling[27] showed that when a normal subject was strongly heated so that there was marked vasodilatation in the forearm skin and a high skin temperature, a fall in the skin temperature of the forearm skin could be obtained by anaesthetizing the cutaneous nerves supplying the area. They interpreted this to mean that a vasodilator influence had been removed by blocking the nerve and, therefore, the skin was supplied by vasodilator nerves. These observations were confirmed[28] and more recently a decrease in flow through the forearm skin on blocking its nerve supply was again shown by the observation that the oxygen saturation of the blood from the skin veins decreased[29].

Other recent evidence has been obtained by Edholm, Fox and Macpherson[30, 31] who based their belief on the facts that (a) during body heating an increase in the rate of blood flow, measured by plethysmography, occurs in the forearm and (b) this increase can be prevented by sympathectomy; (c) the increase occurs entirely in the skin of the forearm; (d) blocking the cutaneous nerves of the forearm produced little change in the total blood flow but prevented an increase in flow during body heating; (e) cutaneous nerve block during the period of body heating causes a decrease in forearm blood flow. They reasoned that, if the rise in skin blood flow could not be brought about by anaesthetization of the cutaneous nerves, the

increase during body heating in the unblocked forearm must have been due to activation of vasodilator fibres to the skin. The decrease in flow produced by nerve block during body heating was the result of blocking the vasodilator fibres.

The vasomotor nerves supplying the skin of the whole limb are obviously intimately concerned with body temperature regulation. Evidence from investigation of the oxygen saturation of blood from veins draining forearm skin and of blood from veins draining the muscles suggests that the vasomotor nerves to the skin of the proximal parts of the limbs do not play a prominent role in blood pressure regulation[19]. The oxygen saturation of blood from the superficial veins did not change while the saturation of blood from the deep veins changed markedly during passive alterations of body posture. However, plethysmographic studies of hand blood flow during passive body tilting does indicate that some vessels in the hand constrict when a subject is passively tilted head up[32] and possibly these are skin vessels. Skin temperature studies on digits have been contradictory.

Summary

The evidence presently available seems to show that (a) in the limbs the blood vessels of the muscles are supplied with sympathetic vasoconstrictor nerves and probably also with sympathetic vasodilator nerves; (b) the skin proximal to the hands (and probably proximal to the feet) is also provided with vasoconstrictor and vasodilator fibres; (c) the skin of the hands and the feet is copiously supplied with vasoconstrictor nerves, but the existence of vasodilator fibres is in doubt. The function of the vasomotor nerves to the muscle vessels is probably related to blood pressure regulation and is not primarily concerned with temperature regulation, while the vasomotor nerves to the skin vessels are intimately concerned with body temperature regulation since the vasoconstrictor nerves are inhibited and the vasodilator nerves are activated by body heating. The vasomotor nerves supplying muscle vessels and mediating reflexes concerned with blood pressure regulation can function independently of the vasomotor nerves responsible for changes in cutaneous vessels in response to changes in body temperature[19].

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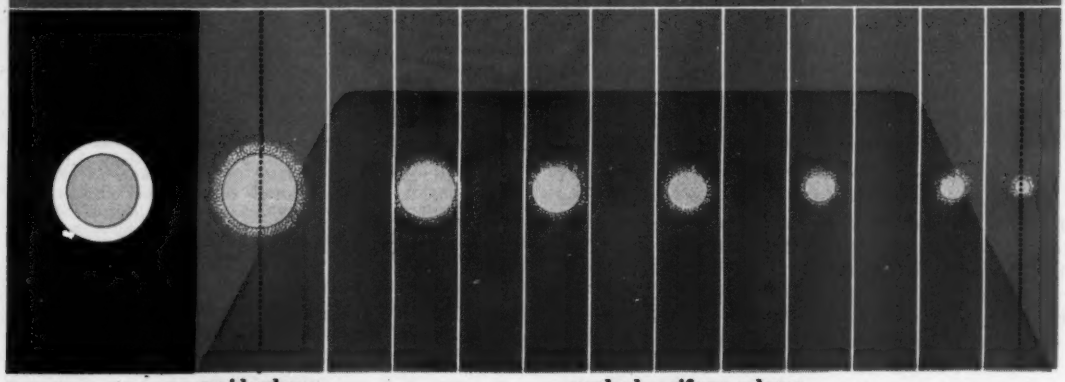

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Editorial

S. Vaisrub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.), F.A.C.P., Editor

The Harvey Tercentenary

June, 1957, marks the tercentenary of the death of William Harvey. This occasion will be commemorated in London at the Royal College of Surgeons by the Harvey Tercentenary Congress during the week June 3rd - June 7th. The full program of the Congress, the main theme of which will be a review of the present knowledge of the circulation, is published elsewhere in this issue.

Although it is not the wont of our provincial publication to publicize international medical events, an understandable exception is made for this occasion due to the exceptional importance of the man whose memory it honors—a man, "whose work" — in the words of Fielding Garrison "has exerted a profounder influence upon modern medicine than that of any other man save Vesalius."

William Harvey belongs in that small select group of men for whom we reserve such epithets as path finders, torch bearers, trail blazers, or more pointedly—makers of history. He has left a mark on the history of medicine, which can be compared with that of Galileo before him on the history of physics or that of Lavoisier after him on the history of chemistry. By virtue of his momentous discovery of the circulation he has joined the ranks of the immortal great.

Along with the ineffable quality of greatness Harvey shares with the great men of history their other characteristic trait—that of being a figure of controversy. In his lifetime he has been misunderstood and doubted by many of his contemporaries. After his death, and the general acceptance of his views, the priority of his discovery has been questioned repeatedly. Attempts to credit Cesalpino with the discovery of the circulation have been persistent enough to be posthumously embarrassing to Harvey's memory.

The controversial aspects of genius, however, are not always confined to disagreements between his protagonists and antagonists. More often than not they manifest themselves in the divergent and sometimes contradictory interpretations of the personality and achievement of the great man by his admirers. If, as often happens, the latter belong to different schools of thought, they end up claiming the great man, as well as acclaiming him. By seizing one aspect of the man and overemphasizing it, they attempt to identify his views with their own. We need only to be reminded of Plato who was "claimed" by no less than six schools of philosophy, or of Buddha, who was "appropriated" by numerous religious sects, to appreciate the extent of the diversity in interpretations of the master by his disciples.

In the case of Harvey the disagreements are focussed not on the substance of his discovery, but on his method of arriving at it. Some historians, in fact, attach more importance to the method than to the discovery in which it culminated. To quote Fielding Garrison: "The discovery of Harvey's work is not so much the discovery of the circulation of the blood as its quantitative or mathematical demonstration." The means appear to overshadow the end.

If the method of Harvey's work is so important, it deserves a closer look. Is the method, as Garrison intimates, merely that of quantitative or mathematical demonstration, of application of measurement to medical science? The answer to this question is neither in the affirmative nor in the negative, for the view is only one of the several differing conceptions of Harvey's method. It is but one of the various interpretations to which the work of this great man has been subjected.

Another conception of Harvey's method, one favored by many physiologists, is, that it is the method of experimental physiology. "Harvey is the founder of the experimental method" states J. A. Ryle in his book "The Background to Modern Science." In support of this view are quoted the experiments that Harvey conducted on animals and humans, experiments involving perfusion, ligation and vivisection. It is to be remembered that he demonstrated the flow of blood toward the heart in the superficial veins of the forearm; it is to be recalled that he demonstrated the effect of a ligature sufficiently tight to obliterate the arterial pulse, as contrasting with that of a loose ligature which, while obstructing the venous return, permited the arterial flow. It is not to be forgotten that these experiments were conducted against the background of an age when medicine was a blend of blind authority, idle speculation and uncouth observation, and that they mark the beginning of a new era of a more fruitful approach to scientific investigation in medicine.

A view diametrically opposite to the above is held by many of the present day biographers of Harvey. They claim that Harvey's experiments were of a limited nature, playing but a small part in the overall picture of his discovery. According to them, reasoning rather than experiment played the dominant role. They regard Harvey as primarily a thinker and an observer. This view is so lucidly expressed by J. Harold Burn in the first chapter of his book on "Function of Autonomic Transmitters" (reviewed elsewhere in this issue) that it is difficult to resist the temptation of quoting from it at length. "The idea of the circulation of the blood"—writes Burn—"probably came to his (Harvey's) mind first, and then he sought evidence to support it. The support provided by the careful and highly informed reasoning drawn from obser-

vation on snakes and fish as well as from studies of the fetal circulation, was perhaps even more convincing than the limited number of experiments which he actually described. He represents one type of scientist who, because of a peculiar intention, is able to sense the weakness of a current conception and to construct in his own mind from various clues, which seem unimportant to others, to explain a problem which interests him. He then proceeds to collect the evidence which will tell him, as it accumulates piece by piece, whether his hypothesis is correct."

Still another interpretation of the means by which the historic end of Harvey's discovery was attained is given by W. Pagel. According to Pagel, (Bulletin History of Medicine XXIV, 1: 71) Harvey was not only an observer, but also a reflective philosopher with a mystic bend, and an Aristotelian background. Leaning towards Aristotle's philosophy of circles and believing in the parallelism between microcosmic and macrocosmic phenomena, he deduced the circular motion of the blood flow in the circuit as but "one instance of a general cosmic law of circles, of periodical processes and circular patterns which reveal a reversion to the point of departure."

Harvey the quantitative computer, Harvey the experimenter, Harvey the thinker, Harvey the mystic—which is the true Harvey? The reader can take his choice. As for the editor, he will buy them all. He will submit that each epithet describes but one facet of Harvey's multi-faceted personality, a personality which in many ways symbolizes the spirit of Science, for, in the words of A. R. Hall ("The Scientific Revolution") "Science is not simply the product of one attitude to nature, of one set of methods of inquiry, or the pursuit of one group of aims. Within it there is room for . . . a greater or less exactitude in observation, and for a considerable latitude in theorization." He will also submit that it is impossible to do full justice to Harvey by confining attention only to his method. A full appreciation must include a reference to his creativity. To quote I. Maclaren Thompson ("William Harvey And His Times," published in this issue)—"After all, knowledge is made not by methods (as Bacon would have it), but by men and women. Harvey had the creative artistic gift that transcends method and leads its possessor to his goal."

Harvey beckons to us across three centuries of time, inviting us to his Tercentenary Congress across three thousand miles of space. May he accept benevolently from those of us who are unable to make the pilgrimage, the tribute of this Editorial and of the Symposium on the Circulation, dedicated to his memory.

Ed.

The Manitoba Heart Foundation

Having paid tribute to the discovery of the circulation, the editorial page now takes leave from the historic past of Cardiology to turn to the future in welcoming the Manitoba Heart Foundation.

A newcomer to this province, the Manitoba Heart Foundation is to be patterned after those of Ontario, British Columbia and Saskatchewan established within the past few years. These provincial bodies are autonomous members of the National Heart Foundation of Canada, which concerns itself with the problems of heart disease at the national level including the raising of funds, integration of research, and the education of both the medical profession and the public in respect to heart disease. The Manitoba Heart Foundation, pursuing similar purposes at a provincial level, will further coordinated effort in research, education and community service, integrated with that of the National Heart Foundation along the lines established by the sister organizations.

Not the least of the tasks of the Foundation is the collection of funds. In this, as in many aspects of its function, the Heart Foundation requires the support of the interested layman. Indeed, the organization is a joint project of the medical profession and the lay public. It is a partnership, and therein lies its strength. Based on common interest in a vital cause, it will prove enduring as well as fruitful. It will also pari passu add another link to the chain of good Public Relations between the Profession and the Public.

Ed.

Obituaries

Dr. Arthur Woods-Hicks

Dr. Arthur Woods-Hicks, 82, died March 12. Born in Mitchell, Ontario, he graduated in 1903 from Trinity Medical College, Toronto. He practised in Halbrite, Shaunavon and Ceylon, Sask. and Radcliffe, Alberta before coming to Winnipeg in 1925. From 1936 to 1951 he practised at Roblin, then returned to Winnipeg. He was a past master at Shaunavon Lodge, A.F. & A.M. His son, a member of the R.C.A.F. was killed in 1941. He is survived by his daughter and two grandchildren.

Dr. Ian Gilhuly

Dr. Ian Gilhuly, 58, died March 25. Born in Selkirk, Manitoba, he graduated M.D. from University of Manitoba in 1925 and practised at Merwin, Sask., Rapid City and Roblin before moving to Minnedosa in 1942. He served in both World wars with the rank of Major in the second. He was a member of the Masonic Order, Rotary and Canadian Legion. He is survived by his widow, a son and a daughter.

Social News

Reported by K. Borthwick-Leslie, M.D.

The big excitement this month of course is political. Congratulations to Dr. Roy W. Richardson, on his nomination as Liberal candidate for Winnipeg South Centre. All the best Roy, in the battle looming up, and that's from a dirty old Tory, too.

♦

Col. Kenneth J. Coates, Virden, Man., has been appointed Command Medical Officer for Prairie Command Headquarters in Winnipeg. Col. Coates has been commandant of the R.C.A.M.C. school, Camp Borden, and is succeeding Col. C. C. Wood, recently retired from the Military.

♦

Dr. and Mrs. J. Hillsman are counting the days until May 22nd, when they take off for the East, to attend Convocation in Montreal where son Dean graduates in Medicine from McGill, then to South Hadley, Mass., where Margaret graduates from Mount Holyoke College, then to New York, where Dr. John attends the American Medical Association convention June 3 - 7.

Happy holiday to all.

♦

Dr. and Mrs. E. K. Vann are enroute also, attending the American Urological Meeting in Pittsburgh and on to Montreal to the Canadian and British Urological Sessions.

♦

Dr. H. Medovy and co-workers are to be congratulated on the organization, under Dr. Lyle McDonald's supervision at the Winnipeg Children's Hospital, of the new "Poison Centre." The Centre will provide information to doctors in the province, on more than 3,000 poisonous substances found in homes and industry, and around the clock emergency service as well. It would seem from the increasing numbers of poisoning in children, that some form of public education is needed.

Perhaps the profession should incorporate such facts into its premarital ("birds and bees") talks.

♦

Helen Tennant, Manchester, Eng., graduate of '48 M.B. D.A. (Diploma of Anaesthesia) has arrived in Winnipeg to become Asst. Anaesthesiologist at Grace Hospital. Welcome to Canada, Dr. Tennant.

♦

Fish story of the month: Dr. Sam Peikoff is the hero. From the depths of the Pacific, off Acapulco, Mexico, he brought to shore a Marlin, 9½ feet long and weighing 110 lbs. If you don't believe it, drop in to his office, where it will be on display shortly. I understand that in the rules of the game, one is not allowed to either give up the struggle or be helped by members of the crew, so poor Sammy nearly had a Coronary before landing the brute. Immediately before, Ruvin Lyons also caught one, but to his chagrin, but better health, his was the one that got away. There must be a moral to this tale; could it be that surgeons can and do catch bigger fish than obstetricians?

♦

The medical stork has not been too active, but congratulations to these proud parents:

Dr. and Mrs. Carey, Clearwater Lake Sanatorium, The Pas, twin boys, Michael John and Murray James, March 14, 1957.

Dr. and Mrs. M. F. McInnes, announce the birth of a daughter, April 4, 1957.

♦

Dr. and Mrs. Robert Cooke (nee Dr. Ruth Grahame) a son, May 3, 1957.

♦

Dr. and Mrs. Wilfred A. Bigelow celebrated their golden wedding anniversary in Brandon, Man., where Dr. Bigelow is the veteran physician and surgeon, and founder of the Bigelow Clinic.

Sincere good wishes to Dr. and Mrs. Bigelow.

♦

Gleaned from the College of Physicians and Surgeons, via the Canadian Doctor, reporting on some of our graduates and also our "imports."

Internal Medicine
Fast, Bernhard, Man. '51, Boston, Mass.
Fyles, Thomas, Man. '49, Winnipeg, Man.
Hughes, J. F. S., Man. '49, St. Boniface, Man.
Varnam, G. S., Man. '51, Winnipeg.
Judge, C.M., Man. '51, St. James, Man.
Judge, David, Man. '51, Edmonton, Alta.
Morris, Jeffrey, Manchester '45, Winnipeg.

Diagnostic Radiology
Fraser, Robert G., Man. '45 Mount Royal, Que.

Surgery
Burns, Chas. M., Man. '51, Winnipeg.
DuVal, Frederic, Man. '50, Winnipeg.
Miller, Jas. A., Western '46 Winnipeg.
Resenfield, Morley, Man. '52, Winnipeg.
Theman, Adolph, Man. '43, Edmonton, Alta.

Ophthalmology
Reed, Howard, Eng. '41, Winnipeg.

Urology
Govan, Duncan, Man. '48, Regina, Sask.
Williams G. D., Man. '50, Willowvale, Ont.

Certification Anaesthesia
Crawford, Wm. J., Belfast '50, Winnipeg.
Drulak, Stephen, McGill '43, Winnipeg.
Hoe, Grant, Man. '50, Edmonton, Alta.
McGarry, Pat, Ireland '46, Winnipeg.
Todd, Thomas, Belfast '49 St. Boniface.

Bacteriology
Wilt, John, Man. '45, Winnipeg.

Physical Medicine
McKinnon, Carl R., Man. '39, Vancouver, B.C.

Psychiatry
Fisher, Kenneth, London '48, Winnipeg.

Public Health
Black, Donald, Man. '34, Kamloops, B.C.
Whitebread, John, Man. '34, Chilliwack, B.C.

Diagnostic Radiology
Ritchie, Gordon, Man. '48, Winnipeg.
Musick Gordon, Man. '45, Willowdale, Ont.

Therapeutic Radiology
Stevenson, John D., Man. '45, Vancouver, B.C.

General Surgery
Janes, Wilbur, Man. '45, Vancouver, B.C.
Miller, James A., Western Ont. '46, Winnipeg.
Resenfield, Morley, Man. '52, Winnipeg.
Shannon, Lloyd, Man. '46, Waycross, G. A.
Davidson, Allan, Man. '50, Toronto, Ont.
Theman, Adolph Man. '43, Edmonton, Alta.
Thorlakson, Robert, Man. '49, Winnipeg.
Lynge, Harold, McGill '49 (Neurosurgery), Winnipeg.

Obstetrics and Gynaecology
Findlay, John, Man. '39, Brandon, Man.
Hopper, Peter, Eng. '42, Winnipeg.
Kohlmeier, A. C., Man. '49, Sarnia, Ont.
Wood, Everett, Man. '48, N. Vancouver, B.C.

Ophthalmology
Fainstein, Saul, Man. '42, Toronto, Ont.
Knowles, Vernon, Man. '51, Saskatoon, Sask.
Mendelson, Jack, Toronto '44, Winnipeg.
Reed, Howard, Eng. '41, Winnipeg.

Otolaryngology
Jacks, Quentin, Man. '40, Vancouver, B.C.
Jackson, James, Edinburgh '45, Winnipeg.
Mazur, Irwin, Man. '42, Winnipeg.
Goodman, Wilfred, Man. '48, Toronto, Ont.

Association Page

Reported by M. T. Macfarland, M.D.

Report of the Economics Committee

The most important part of this report was its recommendation of support for a brief presented by Manitoba Association of Pathologists, regarding Hospital-Pathologist relationships. This is in fact a report of a committee of the Canadian Association of Pathologists. Because of its general interest, it is presented here in full. Also presented is a brief to be submitted to the Manitoba Government dealing with the provision of laboratory services for the Province of Manitoba.

Report of Committee on Hospital Pathologist Relationships, Canadian Association of Pathologists

(A) Preamble

1. Definition

The word "pathology" is used synonymously with "clinical pathology" and includes all branches of laboratory medicine, such as morbid anatomy, bacteriology, biochemistry, hematology, serology, blood banking, etc.

2. Relationship to Medical Care

It is self-evident that, with the present status of medical science, laboratory medicine is of paramount importance in the maintenance of good standards of prevention, diagnosis and scientific treatment of disease. This function will not diminish in the years to come.

3. Historical Background

Twenty-five years ago most hospitals had a very small laboratory or none at all. The few pathologists at that time available were mostly teachers of pathology in university centres and did not practice laboratory medicine as it is known today.

In order to keep pace with advances in medical science, hospitals, on the advice and urging of their medical staffs, have greatly increased the size and scope of their laboratories. In most hospitals this change has been brought about wisely and in consultation with the hospital pathologist.

Along with the demand for increased clinical pathological services, there have been pyramiding costs of hospitalization. In an attempt to secure additional revenues hospitals naturally began to look to these expanding clinical pathological services as fruitful sources.

Thus many hospitals have more or less gradually moved into the field of medical services for which they charge medical fees or the equivalent.

This policy tends to establish a dangerous and invidious precedent. It transfers the ultimate responsibility for medical service to what is primarily a lay organization. It is possible to forsee that, when particularly hard pressed for revenue, a hospital or institution might desire to make the clinical pathological laboratory a paying rather than a progressive department. This would eventually result in deterioration of the calibre of the medical personnel involved, the quality of the work performed and ultimately would be reflected in inferior patient care.

4. Trend in Recent Years

(a) It is the opinion of hospital architects that most hospital laboratories established in the past five years have already outgrown their physical size.

(b) The volume and diversity of laboratory services performed per patient has more than doubled in the past fifteen years. There is every indication that this increase will continue as the practice of medicine continues to develop and expand.

(c) It follows that the size, equipment and personnel of the clinical pathological laboratory depends upon the number and complexity of the services performed rather than on the number of patients investigated.

(d) Hence, the income required by a clinical pathology laboratory must be correlated with the volume and type of service performed as well as the actual number of specimens submitted.

5. Reasons For Submitting These Principles

Whilst, for the most part, the relationships of hospitals and pathologists have been amicable, these relationships have been rather ill-defined and certainly not uniform. Today with the increasing participation of third parties, whether private or Governmental, as carriers of costs of medical services, it is imperative that a clear-cut distinction be made between hospital care and medical care. It is hoped that the following principles may establish a working relationship between hospitals and pathologists, so that clinical pathological services on which good medical care depends will be enhanced and not compromised.

6. Implementation

Because of the changing pattern of hospitalization and medical care both in Canada and elsewhere, now is the time for Canadian pathologists, through the Canadian Medical Association, to meet with the Canadian Hospital Association in order to define clearly the relationship between the pathologist and the hospital.

(B) Principles

1. Status

(a) Pathology is a specialty in the practice of medicine.

(b) The services performed by licensed medical practitioners in this specialty are medical services and not part of hospital care.

2. Policy

(a) It is the aim and policy of the pathologists of Canada to make the highest possible standard

of laboratory medicine generally available throughout the country.

(b) A licensed physician, preferably a certified pathologist, should be in charge of each hospital laboratory.

(c) Pathologists are responsible for the work of the technical laboratory staff.

(d) Pathologists, through the Provincial branches of the Canadian Medical Association, will assume responsibility for setting up standards to ensure the quality and accuracy of pathology as practiced in Canada.

3. Remuneration

(a) In the interests of the public welfare, no corporation or lay body should offer to the public the service of a medical practitioner — which includes pathologists — and charge the patient for those services.

(b) Payment for clinical pathological services should be fundamentally on a fee for service basis whether this payment be made directly by the patient or by a third party.

(c) Fees for the services rendered should provide adequate coverage for all expenses associated with the provision and maintenance of a first class pathological service.

(d) All revenues derived from the clinical pathological services should be applied to these services alone.

(e) Charges for pathological services should be based on a Provincial tariff for such services.

(f) Any billing or charges for pathology services should include the name of the doctor concerned. If deemed advisable there may be included, as a part of the bill, a statement as to what proportion of the charge reverts to the hospital for the non-professional services and facilities which have been provided by the hospital.

(g) Fees and/or charges for pathology services should be paid for as medical and not as hospital services.

The Provision of Laboratory Services for the Province of Manitoba

Introduction

The provision of laboratory diagnostic services is an important component of a program of complete modern medical care. The objectives of modern medical care are two-fold, namely, early diagnosis and early treatment of illness. These result in a reduction in both the duration and severity of illness, providing an economic benefit to the patient which is immeasurable. The importance of diagnostic services in the achievement of these objectives may be measured by the improvement of medical care following their availability. The problem of providing laboratory services to the people of Manitoba is the subject of this presentation.

A laboratory diagnostic service must consist of the following:

(a) Physical facilities.
(b) Technical personnel.
(c) Pathologist's services.

By physical facilities is meant the working space and equipment necessary to carry out laboratory tests. Technical personnel are graduate laboratory technicians who are capable of carrying out laboratory procedures. Pathologist's services are necessary for the supervision of the performance of laboratory tests and their interpretation. The result of a laboratory test that is inaccurate is not only wasteful but may also be dangerous to the life of a patient if it is to provide the basis of treatment. Accuracy of reporting of laboratory tests is thus a measure of all three of the above components; physical facilities must be adequate, technical personnel must be competent, and pathologist supervision and interpretation must be available. Without an adequate standard maintained by the provision of all three of the above, laboratory services cannot fulfill their proper role in medical care.

Present Facilities

Medical centres such as Winnipeg and Brandon, with their large general hospitals have little difficulty in providing diagnostic facilities for practising physicians in these areas. Other areas with smaller general hospitals, however, lack physical facilities for diagnostic procedures because of the low incidence of usage of such procedures and thus higher relative cost, as compared to the above mentioned large general hospitals.

Re-statement of the problem may be made as follows: While large medical centres have laboratory services consistent with their demands, small unit hospitals in rural areas suffer from a lack of diagnostic facilities. How the latter problem may be dealt with is discussed below.

Principle

It is important that practising physicians in Manitoba be encouraged to use laboratory services for the confirmation of clinical diagnoses. The practice of medicine today requires a minimum of laboratory facilities to be available, consistent with the training and experience of the physician practising in the area. Conversely, adequate laboratory facilities will encourage better qualified physicians to settle in areas where these are available.

Since large medical centres are satisfactorily equipped with laboratories, the problem of the rural area and small hospital unit is the subject of the following discussion.

Discussion

It is suggested that the amount and scope of investigation by laboratory examination as a diagnostic measure in either rural or urban areas should be directly proportional to the facilities available in the locality for the treatment of the particular disease.

Elaborate equipment, facilities, highly trained technical personnel, etc., should not be employed to confirm the diagnosis of diseases where the treatment may also require too elaborate equipment and facilities or too highly skilled personnel, all of which is presumed not to be available.

The cost for a diagnosis in terms of technical facilities and personnel in rural areas will amount to more than similar services in larger centres. However, this cost should not be out of proportion to the actual service rendered as compared to the larger centres. Those tests which are infrequent in occurrence and require specialized abilities and facilities to perform are more properly the problem of large centres which can expect a sufficient volume of tests to justify the cost. It is only by this means that a realistic cost basis for laboratory diagnosis can be instituted either in rural or urban areas.

Having excluded a certain group of laboratory tests from rural diagnostic centres, how are they to be so designated? As an initial premise, it is accepted that in the majority of instances laboratory tests are not diagnostic measures in themselves but are merely confirmatory evidence in the substantiation of a well-founded clinical diagnosis on a patient. It is generally conceded that confirmation by specific laboratory tests produces a higher accuracy and thus a better practice of medicine.

From the above it follows that those laboratory tests which should be performed in a given area are the ones which are justified on the basis of volume of demand. This will vary with the district: for example, examination for tuberculosis would be more common in an area with a large Indian population than one composed mainly of other ethnic groups.

The second guiding principle should be based on the availability of competent personnel for the interpretation of the performed laboratory test. A test which is performed without concomitant interpretation is wholly without value. It is not enough merely to remove a tumor from the skin or other organ, since this removal alone does not contribute to the diagnosis. There must also be a pathologist's interpretation. Since the volume of such biopsies does not warrant the full time service of a pathologist, then these tissues, etc., should be forwarded to a centre where such a person is already employed.

Practising physicians are assumed, because of their medical education, to be able to interpret a certain number of laboratory tests without a pathologist's assistance. However, they are not assumed to be able to assess the competence of the performance of a laboratory test by a technician. In this regard then, a pathologist is required to assume some measure of responsibility as to the accuracy of the tests performed. Thus the pathologist plays a dual role in the functioning of a rural diagnostic centre, one interpretive, the other supervisory.

From the above information, the following points may be made:

1. That the specific laboratory tests performed in a given area be representative of the demand for such tests.

2. That the cost of the tests performed be maintained at a level consistent with the cost in large centres, and be arrived at on the basis of sufficient volume of tests performed.

3. That practising physicians be expected to interpret the results of a certain number of adequately performed tests.

4. That practising physicians be not expected to assess the accuracy of laboratory tests on the basis of technician ability; this is a pathologist's function.

5. That pathologists are required to interpret a certain number of laboratory tests, and since pathologists are located in larger centres, that the tests requiring pathologist interpretation should only be performed there.

The problem of accuracy of laboratory testing can be controlled by the two following methods to maintain adequate medical service:

1. By intermittent surveillance on the on-the-spot checking of procedures.

2. By the submission to regional laboratories of control solutions of known values.

The decision as to where laboratory tests should be submitted for interpretation should be guided by two principles:

1. If the patient is to be admitted to a specified large general hospital for diagnosis and treatment, then laboratory tests should be submitted to this same institution.

2. Other than under (1), the practising physician should send his materials for interpretation to any other centre of his own choice, based on his experience of the speed and reliability of the particular centre he has chosen.

. . . . . .

Provision of Pathologists for Diagnostic Services

The proper maintenance of laboratory service requires pathologist supervision. Since diagnostic facilities are widespread in the province the provision of pathologist supervision is difficult. It entails the aggregation of hospital units into areas which will become the responsibility of a pathologist who will be itinerant in his duties. A mailing service can be employed for the sending of unknown samples to be performed in small hospitals as a control measure, and this will avoid considerable time-loss in travel by the pathologist. Personal supervision by a pathologist is not avoided by this means, but frequency of visits may be reduced.

The decision as to the number of pathologists necessary for this supervision will be determined

by the area of country involved and the number of diagnostic units within this area.

It is admitted that at the present time there is:

(1) No division of country points into areas for supervision.

(2) An inadequate supply of pathologists to provide this supervision.

The first problem may be solved empirically by setting up of districts on a provisional basis and assessing results of supervision after a period of time.

The second problem is more difficult and time consuming to solve. Pathologists may be encouraged to migrate to Manitoba through the provision of competitive salaries. Or physicians may be attracted into the specialty of Pathology by providing fellowships or bursaries to enable them to take up this specialty at approved centres. It is important that, if this course is followed, a method of selection of candidates for fellowships be devised to assure that such future specialists will be of a high standard of ability.

Provision of Technicians for Diagnostic Services

One of the major difficulties encountered, even in large centres in providing diagnostic facilities is the provision and maintenance of competent technical personnel.

It is recommended that present hospital facilities be encouraged to expand their output by provincial subsidization of such schools. At the present time training schools graduate sufficient students for local use only. Economically, these schools have little or no incentive to take on more students than they require. For this reason, some form of subsidization must be available to encourage training centres to accept more students than they personally require.

Implementation of Principles

A. Rural Areas

On the basis of experience, it has been found that a hospital with a complement of 120-150 beds having an occupancy rate of 85-100% may require the services of a full time pathologist. Conjointly, to provide adequate service for this same hospital in terms of laboratory tests there should be approximately five graduate laboratory technicians.

If the hospital is smaller in size than the above figure, the ratio of laboratory technicians to bed number should be kept similar, that is, one per 25-30 beds. If the laboratory technician has other duties to perform, such as X-Ray work, then the number of hospital beds she should be responsible for should be decreased by 30 to 50%; that is, one technician to approximately 15 to 20 beds. At 100% bed occupancy, with an average patient stay of ten days, one technician can provide service for approximately 720 patients per year in terms of laboratory work alone, or 400-500 patients as a combined X-Ray laboratory technician.

In the experience of a large hospital, each patient has performed upon him or her an average of six individual laboratory tests. From this, one technician should be responsible for the production of six tests on each of 720 patients per year or approximately 5,000 laboratory tests per year.

With regard to pathologists, one such person could supervise the work and interpret the material from four 30-50 bed hospitals if they were within commuting distance.

In summary, in rural areas where small unit hospitals are desired, it is felt that these hospitals should be of a size to merit the full employment of a laboratory technician. Hospital size should be a minimum of 15 to 20 beds if the technician is to do X-Ray as well as laboratory work, or 30 beds if laboratory work only is required. Hospital size smaller than this capacity would probably be uneconomic and wasteful in terms of technician time and ability.

B. Urban Areas

Physicians practising in large cities desire laboratory facilities of an order similar to that provided physicians in rural areas. Admittedly laboratory facilities within large hospitals are adequate, but since the major part of medical practice takes place within doctors' offices, then this medical practice should be supplemented by a minimum of laboratory tests. It is recommended that the tests that should and can be employed in doctors' offices are those that are within the scope of practising physician interpretation. Other tests than these should be submitted to the laboratory facilities of large hospitals for pathologist interpretation.

The purpose of providing a minimum of laboratory diagnostic aids to physicians within their offices is to avoid costly time delays in the diagnosis and treatment of ambulant patients. If a physician's practice is insufficient to justify the employment of a full time laboratory technician, then a group of physicians may combine their facilities to employ a technician.

However, it must be stressed that merely placing a technician within a laboratory does not guarantee adequate and accurate service. It is mandatory that a system of surveillance and checking of routine procedures within the laboratory be carried out. This is properly the problem of a committee of pathologists or other specialists qualified to ascertain the adequacy and accuracy of the tests. Laboratory procedures as now performed in the majority of private doctors' offices do not have any method for controlling the accuracy of tests performed.

Final Summary

1. Provision of laboratory facilities for physicians in rural or urban areas is a necessary component of the practice of modern medicine.

2. Hospital size should approximate 20 to 30 beds to fully utilize the services of one technician.

3. Hospital size of 120-150 beds or its equivalent

in smaller units is necessary for the employment of one pathologist.

4. One laboratory technician should be capable of providing service to from 500-700 patients per year or to perform approximately 5,000 laboratory tests per year.

5. Specialist surveillance of laboratory efficiency is a necessary part of the adequate functioning of a diagnostic service, either rural or urban.

6. One pathologist is capable of directing the work of approximately five graduate laboratory technicians.

7. Deficiencies in diagnostic services, both rural and urban, are described and means have been suggested for their correction.

Annual Meeting
Program Committee Notice

The Annual Meeting of the Manitoba Medical Association will be held at the Royal Alexandra Hotel, October 15th - 18th, 1957.

Members who have papers which they desire to present are asked to forward details to the Scientific Program Committee, 604 Medical Arts Building, 404 Graham Avenue, Winnipeg 1, prior to May 20th.

Brandon and District Medical Association

A meeting of the Brandon and District Medical Association was convened at 5.00 p.m. on March 8th, 1957, at the Prince Edward Hotel, Brandon, with the President Dr. G. T. McNeill, Carberry, in the chair.

The minutes of the previous meeting were read by the Secretary, Dr. H. McIntyre.

Items discussed were:

1. Payment for report forms by Manitoba Hospital Service Association.
2. Medical examination of immigrants.
3. Medico-Legal Society.

The following were present:

Brandon: Dr. J. B. Baker, Dr. F. J. Burgess, Dr. P. Cole, Dr. R. P. Cromarty, Dr. J. A. Findlay, Dr. F. Fjeldsted, Dr. W. P. Hirsch, Dr. N. C. Horne, Dr. D. L. Johnson, Dr. M. Kozakiewicz, Dr. A. P. Lapko, Dr. J. M. Matheson, Dr. R. O. McDiarmid, Dr. H. M. McIntyre, Dr. J. H. Scott, Dr. H. S. Sharpe, Dr. V. J. Sharpe, Dr. E. J. Skafel, Dr. R. H. Sykes.

Carberry: Dr. G. T. McNeill.

Kenton: Dr. W. K. Hames.

Neepawa: Dr. G. Lambertsen.

Oak River: Dr. J. D. McMillan.

Shilo: Dr. B. D. Morgan (Major).

Winnipeg: Mr. G. L. Cousley, Q.C., Dr. D. J. Fraser, Dr. M. T. Macfarland.

Reception and dinner were provided by the Workmen's Compensation Board and greetings were extended by the Commissioner, Mr. G. L. Cousley, Q.C. Following the dinner there was a good discussion of medical problems which arise in treatment of cases which may be the responsibility of the Board, led by Dr. D. J. Fraser.

While the business session was in progress the ladies were entertained by a European travelogue presented by Miss Ross.

The second session of the meeting was held in the classroom, Brandon General Hospital on Saturday morning, March 9th, 1957 when a clinical session was held.

In attendance were:

Brandon: Dr. J. B. Baker, Dr. P. Cole, Dr. R. P. Cromarty, Dr. J. A. Findlay, Dr. F. Fjeldsted, Dr. W. P. Hirsch, Dr. D. L. Johnson, Dr. A. P. Lapko, Dr. J. M. Matheson, Dr. R. O. McDiarmid, Dr. H. M. McIntyre, Dr. A. H. Povah, Dr. F. J. Purdie, Dr. J. E. Rowlands, Dr. Sharpe, Dr. E. J. Skafel, Dr. R. H. Sykes, Dr. W. Shahariw.

Carberry: Dr. G. T. McNeill.

Deloraine: Dr. W. Malyska.

Hamiota: Dr. J. E. Hudson.

Killarney: Dr. G. E. Dow.

Winnipeg: Dr. D. J. Fraser, Dr. M. T. Macfarland, Dr. R. F. M. Myers.

Dr. J. M. Matheson presented an interesting case of Lymphosarcoma in a patient previously operated on for Hyperthyroidism.

Dr. D. L. Johnson presented a paper on the subject of "Leukemia."

In the absence of Doctors J. P. Gemmell and I. Israels, Dr. R. F. M. Myers discussed Dr. Johnson's paper.

Dr. J. E. Hudson, President, Manitoba Medical Association, discussed several matters with which the Executive and other Committees are now concerned.

Following lunch Dr. Hudson set off for Hamiota by plane to attend waiting patients.

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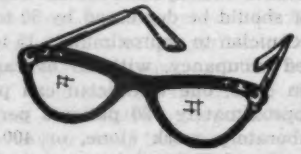

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Retirement Savings Plan

Letter Re Pension Plan

A letter sent out by the Manitoba Medical Association under date of March 19th, 1957, outlining the pension possibilities by your Pension Committee is being followed up and we are publishing the submission by The Canadian Medical Association in this issue.

This will be followed by the plans as applied to the Manitoba Medical Association and the Manitoba Medical Service.

We would greatly appreciate it if the members would study these plans and write to your Committee any criticism or improvement that would help us to arrive at the best plan for the doctors.

Canadian Medical Association Report on Proposed Registered Retirement Savings Plan

The proposed legislation granting income tax deferment on contributions to retirement savings plans establishes only two principal methods of doing so. These two methods may be roughly classified as "insured" and "trusteed." It may be assumed that some variations in these methods and/or a combination of them will be acceptable to the tax authorities.

The "insured" method involves entering into a contract with an insurer who, in return for an annual premium, undertakes to pay to the depositor a stated amount annually commencing at a pre-determined date and throughout his remaining lifetime. Such contracts call for a fixed amount to be paid periodically, and failure to make the payment would result in penalties being incurred. This offers the safest method of providing for an income in the future; provided the premiums are paid as arranged under the contract, the continuance of the payments after the endowment date are as certain as anything can be. Canadian insurance companies have the very highest reputation for meeting their obligations.

There are certain points that should be considered in the use of this method exclusively. One is the rigidity of the premium payments which are, in effect, the purchase price. The depositor is obligated to meet a fixed periodic commitment, and failure to do so subjects him to possible loss. The amount of pension to be ultimately payable, and the date the payments commence are also determined at the time of making the contract. The effect of this is to impose somewhat rigid restrictions which could become a burden or would not satisfactorily meet future changing conditions. It may result in, what seemed at its inception to be a satisfactory income, being quite inadequate at the time it is received.

During recent years, there has been considerable public interest in the "variable annuity" as opposed to the more conventional type of annuity contract which provides a fixed dollar income. The essential feature of the "variable annuity" is that the amount payable to the annuitant may be changed from year to year to reflect gains or losses in especially the investment experience of the insurer. Where this type of annuity is used, the premiums are largely, if not entirely, invested in common stocks and it is reasonable to assume that there will be variations in their net worth from time to time, thereby producing changes in the amount of the annuity payments. It will be seen that the advantages, if any, of the "variable annuity" in relation to the fixed income annuity depend entirely on the relative advantages of equity vs. fixed dollar investments, and in effect, the "variable annuity" is a device to pass along the gains or losses of the equity investments.

The experience with this type of contract is as yet somewhat limited. Since its inception, subsequent to the last war, it has been operating in an economy most favourable to its successful development. An alternative to this is suggested hereunder, in which protection is given against inflation during the accumulation period rather than during the period of pension payments.

The "trusteed" method involves the depositing of monies by each member in a trust company, pursuant to the terms of an agreement which it is suggested could most advantageously be made by the Canadian Medical Association. Under such agreement, amounts paid over either directly or indirectly by members of the Association would be held upon a Trust, the terms and conditions of which would conform to the proposed legislation and regulations, if any, made thereunder.

The monies so held would be invested by the trust company under wide powers granted to them. The depositor could have a choice as to the type of investment in which all or a part of his deposits could be applied. For example, it could be invested in

(i) a fund consisting solely of trustee investments, i.e. government, municipal and provincial bonds, etc.,

(ii) a fund consisting of high grade commercial securities, debentures, mortgages, guaranteed certificates, etc.

(iii) a fund consisting of selected common stocks having earnings, growth or both.

(iv) any combination of the above.

The monies so held and the accumulations thereon would, at or prior to the time when the depositor attains his 70th birthday, be used to purchase from an insurer at the best price then available an annuity contract, the payments under

which would commence immediately. At that time the depositor is in a much better position to select the terms of the contract which can be suited to the circumstances as he then knows them.

A substitute for this proposal is the use of a Deposit Administration (D.A.) Contract with an insurer. This contract provides that monies deposited shall be held and invested by the insurance company with a guaranteed rate of interest. At endowment, the share of any depositor is removed from the account and applied as the premium on a contract to provide such amount of pension as can be purchased from the insurer at the rates then in effect. This method closely approximates the use of a trusteed fund, the more important differences being:

1. The type of securities in which a depositor's share is invested cannot be selected.

2. The "full" benefit of higher earnings or capital appreciation is not received by the depositor.

3. The ultimate insurer is pre-determined.

There is no reason for assuming that an insurance company is a better or safer investor than a trust company. The essential difference is that insurance companies are restricted by law to both the proportion and type of investment which can be made. There is no limitation to the investment field of a trust company; it can cover that to which insurance companies are restricted as well as the remainder of the whole field—it is limited only by good judgment.

An essential requirement is that the money once deposited must remain there until received either as a death benefit or in the form of an annuity to the depositor or his wife. If received in any other way, it is subject to a severe tax. In other words, it is "locked in" but it accumulates free of tax. It is, in essence, pure savings and if so regarded, the contributions should be dealt with as would ordinary savings under a sound investment scheme. This, of course, is to attain the greatest increment and return consistent with safety. But if applied as part of the savings element in a life insurance contract, there would be no tax deferment. The alternative would thus seem to be a sound investment program.

The trusteed method herein proposed is one that can be instituted with a minimum of work or delay. The cost of initiating it is negligible. It involves no contract which might become onerous, and can be altered or amended at any time. In effect, it permits the benefits of the proposed legislation to be immediately available with enough flexibility to make changes should experience show these to be necessary or desirable.

A somewhat broader view of this scheme would be comparable to what has been done by the members of the teaching profession. Following an early attempt to fund teachers' pensions, (teachers including those in a recognized university in the United States, Canada and Newfoundland) it was found that the cost was such that a fully contributory basis was necessary. In order to facilitate this, a life insurance company was incorporated under the laws of the State of New York under the name of the "Teachers' Insurance and Annuity Association" (T.I.A.A.). This organization sold life insurance and annuities only to persons who were on the teaching staff of United States and Canadian Universities. It was a non-profit organization and its records to date indicate that it has been quite successful in meeting the needs of the class for which it was created. It was this organization that developed the "variable annuity." The overall result has been to enable eligible teachers to purchase life insurance and annuity contracts at favourable rates.

Such a scheme is an enlargement of the trusteed pension fund which is the medium through which a substantial number of larger employee pension plans are founded. It is mentioned here as something to be considered should there be a large enough cohesive group which would be willing to participate in such a scheme. This, of course, involves a separate self-supporting company, the costs of which are reflected by the premiums charged.

In the following pages, we outline the important operative provisions of a suggested contract with a trust company. There has been no consultation with a trust company as to these terms and, therefore, it cannot be said if, as drafted, they would be acceptable. It is not intended to be final but merely an indication of what is required and a basis upon which a contract of similar intent can be negotiated.

AGREEMENT made as of the ..... day of ..... A.D. 1957.

BETWEEN:

Canadian Medical Association (hereinafter called the "Association")

AND:

The ..... Trust Company (hereinafter called the "Trustee").

WHEREAS there has been enacted by the Parliament of Canada certain legislation giving favourable tax consideration to contributions to Registered Retirement Savings Plans, and

WHEREAS the Association desires that its members have available for them a Registered Retirement Savings Plan to which contributions can be made, and

WHEREAS the Trustee is desirous of establishing such a Registered Retirement Plan for the benefit of members of the Association.

NOW, THEREFORE, the parties hereto agree as follows:

1. The Trustee shall accept from time to time deposits from the members of the Association. Any such deposits shall be not less than One Hundred ($100.00) Dollars, but may be any multiple thereof,

and shall be sent to the office of the Trustee at the City of Toronto.

2. The Trustee shall receive and hold the deposits from time to time upon the following trusts:

(a) To invest and reinvest the amount of such deposit, or any portion thereof, in such investments as may seem suitable to the Trustee.

(b) To hold the property arising from the investments of the deposits and to collect and hold the earnings including interest, dividends or accretions thereto, and to credit each depositor with a proportionate share of such earnings or accretions.

(c) To reinvest such earnings or accretions and deal with them in the same manner as deposits by a member.

3. The Trustee shall not, except as otherwise provided for herein, refund or pay to any member any part of any deposit received from him or of the earnings or accretions thereto; except that in the event of death of a member, the amount at the credit of such member shall be dealt with as required by the member either by directions to the Trustee under Article ..... hereof or by Will.

4. At or prior to the 70th birthday of the depositor, the Trustee shall apply the amount of the Fund to which the depositor is entitled as the premium on an annuity contract and under the terms of which the depositor must be the primary beneficiary on a life contingency basis. Such contract shall not contain any provisions which are contrary to any provision of the Income Tax Act or regulations made thereunder as it or they may be in force and effect from time to time. The assignment and surrender of such contract shall be in full satisfaction of his rights under this plan, and further deposits shall not be accepted by the Trustee.

5. Any member making a deposit shall agree to and be governed by the terms of this Agreement as it shall be in force and effect from time to time. The Association agrees that it shall inform its members of the terms of this Agreement, and shall do all things necessary to establish and maintain this Agreement and the fund or funds established in connection therewith as a Registered Retirement Savings Scheme under the Income Tax Act.

6. Any amount paid over to the Trustee by a member shall be irrevocably subject to the terms and conditions established under this Agreement, and under no circumstances or conditions shall the Trustee do anything which would operate to deprive this Agreement or the Fund established hereunder of its status as a Registered Retirement Savings Scheme under the Income Tax Act.

7. The terms of this Trust are provisional only, and are subject to such changes as may be required to conform to the terms of any legislation relating to the establishment of a Registered Retirement Savings Plan, or any regulations thereunder. It is agreed between the parties hereto that such changes will be made as soon as such legislation has been passed or regulations made.

8. The Trustee shall have power to deal with the property of the Fund or of that part of the property of the Fund to which any member may be entitled in any manner deemed necessary or desirable to accomplish the purposes of this Trust. In particular, and not in limitation of any general power, the Trustee shall,

(a) sell or exchange,
(b) vote stocks and give proxies, etc.,
(c) execute transfers,
(d) register investments in nominee,
(e) retain a portion in cash,
(f) borrow against the Fund,
(g) employ counsel, agents, etc.,
(h) keep proper records,
(i) incur no liability except by neglect.

9. The fees and expenses of the Trustee as regards the Fund as a whole shall be ascertained by a memorandum in writing and agreed upon by the Association and the Trustee. Such fees shall be subject to review from time to time and as constituted shall be and form part of this Agreement. Any fees or charges for services to a particular depositor shall be paid by such depositor to the Trustee.

10. The property of the Fund shall be held by the Trustee for the benefit of the depositors, and to be applied only as provided herein. The Trustee shall not accept or act upon any assignment or encumbrance, or deal with the property or interest of any depositor other than under the terms of this Trust except as otherwise required by law.

11. This Trust shall continue during the lifetime of any descendant of His Late Majesty King George VI now living and twenty-one years thereafter, less one day, or for such further period as may be lawful.

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Lindsay, J. R.: The Practical Management of Dizziness, California Med. 83:193 (Sept.) 1955.

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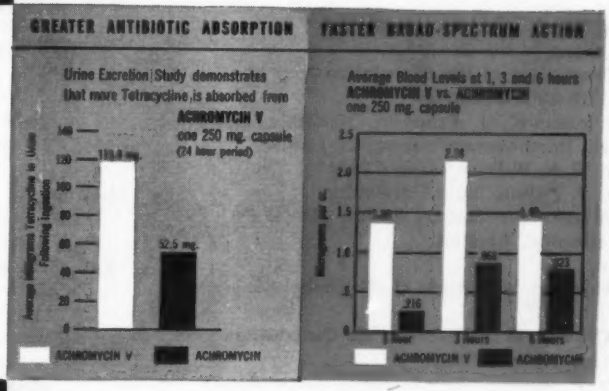

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Harvey Tercentenary Congress 1957
June 3rd - June 7th
At The Royal College of Surgeons, London

The Tercentenary of the death of William Harvey (1578-1657) will be commemorated by an International Congress on the Circulation. The main theme will be:

"A Review of the Present Knowledge of the Circulation"

Monday, June 3rd

Morning

Chairman: The President.

9.30 Opening of the Congress.

"Knowledge of the Circulation from the 17th-20th Centuries."

Professor K. J. Franklin, Medical College, St. Bartholomew's Hospital, London.
Dr. F. A. Willius, Mayo Clinic.
Dr. J. Fulton, Yale University.
Professor Sir Charles Dodds, Middlesex Hospital, London.

Afternoon

2.00 Chairman: Professor G. W. Pickering, Oxford.

"The role of the heart in the Circulation."

Dr. L. Katz, Chicago.
Dr. P. Wood, National Heart Hospital, London.
Professor K. Matthes, Heidelberg.
Dr. Silvio Weidmann, Berne.

Tuesday, June 4th

Morning

Chairman: Sir Clement Price-Thomas, Westminster Hospital, London.

9.30 **"The results of cardiac surgery."**

Sir Russell Brock, Guy's Hospital, London.
Professor G. d'Allaines, Paris.
Professor C. Crafoord, Stockholm.
Dr. Maurice Campbell, Guy's Hospital, London.

Afternoon

2.00 Chairman: Dr. C. S. Beck, Cleveland.

"The coronary Circulation."

Dr. D. E. Gregg, Washington.

Wednesday, June 5th

Morning

Chairman: Professor J. McMichael, Postgraduate Medical School, London.

9.30 **"The pulmonary Circulation."**

Dr. A. Cournand, New York.
Professor C. V. Harrison, Postgraduate Medical School, London.
Dr. S. Radner, Lund, Sweden.

Afternoon

2.00 **"The foetal Circulation."**

Dr. G. S. Dawes, Nuffield Institute for Medical Research, Oxford.

Thursday, June 6th

Chairman: Dr. Macdonald Critchley, King's College Hospital, London.

Morning

9.30 **"The cerebral Circulation."**

Dr. S. Kety, National Institute of Health, Bethesda.
Professor Th. Alajouanine, Paris.
Dr. E. H. Botterell, Toronto.

Afternoon

2.00 Chairman: Professor Sir James Learmonth, Edinburgh.

"The splanchnic Circulation."

Dr. S. E. Bradley, New York.
Dr. S. Sherlock, Postgraduate Medical School, London.
Professor R. Milnes Walker, Bristol.

Friday, June 7th

Morning

Chairman: Professor A. Kekwick, Middlesex Hospital, London.

9.30 **"The peripheral Circulation."**

"Circulation through the limbs": Professor H. Barcroft, St. Thomas' Hospital, London.
"Vascular innervation": Professor W. D. M. Paton, Royal College of Surgeons, London.
"Pathology of vessels": Professor J. H. Dible, Postgraduate Medical School, London.
"Surgery of occlusive arterial disease": Professor C. G. Rob, St. Mary's Hospital, London.

It will be followed by a week-end Conference on the more personal and biographical aspects of William Harvey's life at his birthplace—Folkestone, Kent—on Saturday, June 8th, 1857:

Saturday, June 8th

Chairman: Sir Geoffrey Keynes.

10.30 Harvey's birthplace: Professor T. Hare, London.
Harvey at Cambridge: Professor Sir Lionel Whitby, Cambridge.
Harvey at Padua: Professor A. P. Cawadias, London.
Harvey and French Medicine: Professor L. Chauvois, Paris.
Harvey the Scientist: Dr. W. R. Bett.

Afternoon

Visit to Canterbury Cathedral.

Evening

Civic Reception.

Registration Fees: £10 0s. 0d. sterling for full Membership to both the Scientific programme and Social functions. Alternative enrolment fee of £1 0s. 0d. sterling daily for Scientific Sessions only. Further details and application for membership from:

Congress Secretary, 11 Chandos Street, Cavendish Square, London, W.1

Department of Health and Public Welfare

Comparisons Communicable Diseases — Manitoba (Whites and Indians)

DISEASES	1957 Feb. 24 to Mar. 23,'57	1957 Jan. 27 to Feb. 23,'57	1956 Feb. 26 to Mar. 24,'56	1956 Jan. 29 to Feb. 25,'56	Total Jan. 1 to Mar. 23,'57	Total Jan. 1 to Mar. 24,'56
Anterior Poliomyelitis	....	....	2	1	2	3
Chickenpox	123	132	86	126	344	296
Diphtheria	9	3	....	....	17	....
Diarrhoea and Enteritis, under 1 year	17	19	9	15	36	28
Diphtheria Carriers	4	2	....	....	9	....
Dysentery—Amoebic	....	....	....	....	....	....
Dysentery—Bacillary	....	....	1	3	....	5
Erysipelas	3	1	2	1	4	6
Encephalitis	....	....	....	....	....	....
Influenza	31	3	9	14	34	28
Measles	519	555	210	256	1310	604
Measles—German	22	23	31	36	54	72
Meningococcal Meningitis	....	1	....	1	1	2
Mumps	113	91	196	171	241	479
Ophthalmia Neonatorum	....	....	....	....	....	....
Pneumonia, Lobar	....	....	....	....	....	....
Puerperal Fever	....	....	....	1	....	1
Scarlet Fever	12	33	21	18	49	57
Septic Sore Throat	....	1	....	4	1	4
Smallpox	....	....	....	....	....	....
Tetanus	....	1	....	....	1	....
Trachoma	....	....	....	....	....	....
Tuberculosis	41	31	45	27	90	84
Typhoid Fever	....	1	....	....	1	....
Typhoid Paratyphoid	....	....	....	....	....	1
Typhoid Carriers	1	....	....	....	1	....
Undulant Fever	....	....	1	1	....	2
Whooping Cough	20	35	28	36	67	73
Gonorrhoea	77	61	123	106	202	328
Syphilis	7	10	8	7	22	18
Jaundice, Infectious	117	46	25	33	181	70

Four Week Period February 24 to March 23, 1957

DISEASES (White Cases Only) *Approximate population	*850,000 Manitoba	*880,665 Saskatchewan	*5,404,933 Ontario	*2,953,000 Minnesota
Poliomyelitis	....	....	1	1
Chickenpox	123	29	2796	....
Diarrhoea and Enteritis under 1 yr.	17	....	....	....
Diphtheria	9	....	3	4
Diphtheria Carriers	4	....	....	....
Dysentery—Amoebic	....	....	....	....
Dysentery—Bacillary	....	5	9	3
Encephalitis Epidemica	....	....	....	....
Erysipelas	3	....	4	....
Influenza	31	....	3	102
Jaundice, Infectious	117	112	58	24
Measles	519	57	2072	1751
German Measles	22	4	257	....
Meningitis Meningococcal	....	1	5	....
Mumps	113	35	1320	....
Psittacosis	....	....	....	....
Pertussis	....	....	....	....
Puerperal Fever	....	....	....	....
Scarlet Fever	12	5	285	90
Septic Sore Throat	....	4	4	48
Smallpox	....	....	....	....
Tetanus	....	....	....	....
Trachoma	....	....	....	....
Tuberculosis	41	31	86	99
Typhoid Fever	....	....	3	....
Typh. Para-Typhoid	....	....	1	....
Typhoid Carrier	1	....	....	....
Undulant Fever	....	....	3	....
Whooping Cough	20	....	51	167
Gonorrhoea	77	....	101	....
Syphilis	7	....	25	....

DEATHS FROM REPORTABLE DISEASES

March, 1957

Urban — Cancer, 54; Jaundice (Infectious), 2; Measles, 1; Pneumonia, Lobar (490), 1; Pneumonia (other forms), 10; Septicaemia & Pyaemia, 1; Syphilis, 2; Tuberculosis, 2. Other deaths under 1 year, 24. Other deaths over 1 year, 220. Stillbirths, 13. Total, 330.

Rural — Cancer, 23; Diarrhoea & Enteritis, 4; Measles, 2; Pneumonia, Lobar (490), 2; Pneumonia (other forms), 6; Septic sore throat, 1; Septicaemia & Pyaemia, 1; Tuberculosis, 3; Whooping cough, 2. Other deaths under 1 year, 9. Other deaths over 1 year, 159. Stillbirths, 6. Total, 218.

Indians—Diarrhoea and Enteritis, 1; Influenza, 1; Pneumonia (other forms), 1; Tuberculosis, 1; Chickenpox, 1. Other deaths under 1 year, 3. Other deaths over 1 year, 1. Stillbirths, 1. Total, 10.

♦

Comment

Diphtheria — Nine cases and four carriers were reported early in March but the last onset date of any reported case was March 13.

Measles—This has been very prevalent since the beginning of the year, more than double the number in the same period last year.

Typhoid Carrier—This carrier was discovered after investigating the source of the disease in a three-year-old. The carrier gave a history of having typhoid fever in 1910.

Abstracts from the Literature

Relation of the Postcommissurotomy Syndrome to the Rheumatic State. Daniel L. Larson. Circulation 15: 203-209, Feb. 1957.

This report is designed to present evidence that postcommissurotomy syndrome is probably not due to rheumatic activity, and a comparison is drawn between patients who did and those who did not sustain an attack in the postoperative phase. Thirty-seven percent of 137 patients surviving mitral commissurotomy developed chest pain, fever and elevated sedimentation rate three or more weeks after operation. None of the cases demonstrated group A hemolytic streptococci, circulating antibodies to streptococcal antigens, or elongation of the P-R interval on the electrocardiogram. Postcommissurotomy syndrome was seen more than twice as often in women as in men.

The history of rheumatic fever, rheumatic activity before operation, and seasonal incidence were judged to have no significant part in precipitation of the syndrome. The rate of post operative ambulation and post operative prophylaxis were managed along the same lines in both groups of patients. Eighteen patients developed an attack while taking penicillin, and 11 while on prophylactic sulfadiazine. Three cases sustained recurrences while on maintenance doses of prednisone.

The clinical course of the syndrome was variable, with a prolonged course in some, despite therapy considered optimal for treatment of rheumatic fever. It is emphasized that subacute bacterial endocarditis, thrombo-embolic disease, drug sensitivity, and idiopathic pericarditis must be differentiated from the syndrome. Aschoff nodules in atrial appendages were not significantly more common in the affected group. Seven percent of patients with an uncomplicated course showed an anaemia, while 46% of those having the syndrome had a drop in haemoglobin.

A complication indistinguishable from the postcommissurotomy syndrome has been observed in patients undergoing chest surgery for condition other than rheumatic heart disease. Patients sustaining the syndrome showed no additional cardiac damage, and even after several attacks had as good a functional result as those with an uncomplicated course.

Typical rheumatic fever may occur in the post operative period but it is concluded that the majority of postcommissurotomy syndromes are not exacerbations of rheumatic activity.

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Letter to The Editor

March 15, 1957.

Sir:

My elected term as a member of the Board of Trustees of the Manitoba Medical Service has come to an end.

Through you, Sir, I would like to express my appreciation to the other members of the Board for the pleasure of joining in with their deliberations. My period of service on the Board has assured me that the business of the Manitoba Medical Service is in the hands of a group of citizens, medical and non-medical, whose deliberations are in the best traditions of our profession, namely conscientious service to the public.

Further I would like to express sincere appreciation of the work and the wisdom of the executive director. In this field of exploration in prepayment health plans he has given devoted and imaginative guidance to our deliberations. He has performed his high executive office with understanding, with kindness and with skill.

I leave assured that the important economic function that M.M.S. serves both to the profession and to the public could not be in better hands.

Sincerely yours,

A. A. Klass, M.D.

Dear Sir:

Doctor R. E. Helgason, M.D., of Glenboro, Man., has been awarded a scholarship by the Manitoba Chapter of the College of General Practice. The presentation of the scholarship to Doctor Helgason was made at the annual Valentine Dinner held at the Royal Alexandra Hotel in February, 1957. Doctor Helgason received his Bachelor of Arts degree from the University of Saskatchewan in 1941, and his medical degree from the University of Manitoba in 1945. Following his graduation he served with the R.C.A.M.C. Doctor Helgason is now practicing in Glenboro, Manitoba, where he conducts a very active practice. However, he has found time to do post-graduate work at the University of Minnesota and also at the Cook County Graduate School of Medicine. Doctor Helgason has expressed his intention to use this scholarship to do further post-graduate studies in Obstetrics and Gynecology. Doctor Helgason was married in 1945 to Miss Margaret Johnson of Glenboro, Manitoba. They have three children. Doctor Helgason likes photography and curling.

The Manitoba Chapter of the College of General Practice are happy in their choice in Doctor Helgason and wish him every success in the future.

Yours truly,

R. A. Jacques, M.D.

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